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PRACTICAL OBSERVATIONS

ON CERTAIN

DISEASES OF THE CHEST,

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PRACTICAL OBSERVATIONS

ON CERTAIN

DISEASES OF THE CHEST,

AND ON THE

PRINCIPLES OF AUSCULTATION.

BY

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LONDON:
JOHN CHURCHILL, PRINCES STREET, SOHO.
MDCCCXLVIII.

PRINTED BY C. AND J. ADLARD,
BARTHOLOMEW CLOSE.

TO

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AND MEMBER OF SEVERAL LEARNED SOCIETIES BOTH
FRENCH AND FOREIGN,

In grateful acknowledgment of the advantages derived from the study of his Works, and from his valuable counsel and instruction;

AND TO

WILLIAM BOWMAN, F.R.S.,

ASSISTANT SURGEON TO THE KING'S COLLEGE AND ROYAL OPHTHALMIC
HOSPITALS, LONDON; CORRESPONDING MEMBER OF THE PHILOMATHIC SOCIETY
OF PARIS, AND OF THE ROYAL ACADEMY OF SCIENCES OF TURIN;

AND

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The Author's valued Friends and former Clinical Pupils:

THIS WORK

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PREFACE.

THE method of investigating Diseases of the Chest has been greatly modified by the discovery of auscultation; since the introduction of which numerous facts have been collected, and some rules for diagnosis and treatment have been deduced from them. Certain of these deductions are universally admitted to be sound, but others require further confirmation before they can be considered as firmly established. There are still, however, some forms of disease, for the detection or treatment of which no rules have as yet been laid down.

Additional facts, therefore, are required, which may lead to the further establishment of sound general principles, and which may serve to prove the truth or fallacy of those which have been already proposed. As these facts can only be furnished by those who have had favorable opportunities for investigating disease, it is very desirable that such persons should record and publish their observations, even should they be compelled by a want of leisure or ability to leave to others the task of eliminating from them general truths by philosophical induction.

But even were the principles of medical science firmly established on all points, collections of accurately reported cases would still possess considerable value. For in proportion as the rules for diagnosis and treatment become fixed on a firm basis, so is there a danger of the young practitioner endeavouring to discover and treat disease by general rules alone, irrespective of that careful study of the peculiarities of each individual case, which forms a leading feature in the practice of the experienced physician. This danger may, in some degree, be avoided, by a frequent reference to recorded cases, for the purpose of comparing them with those under treatment. It is partly on this account that the works of Abercrombie, Andral, and Louis have always been held in such high estimation by practical men.

Influenced by these considerations, the Author of the following pages has ventured to submit them to the profession, although they contain little more than a faithful report of facts observed in private and hospital practice, and also in a long course of gratuitous attendance on the lower orders.

Many medical gentlemen have also, with great kindness, brought under the Author's notice such cases as they thought would assist him in his researches, to whom he takes this opportunity of returning his grateful acknowledgments.

In almost every one of the cases here recorded, the symptoms during life were witnessed and noted down by the Author; and, in the event of death occurring, he was either present at the examination of the body, or inspected the diseased parts after their removal. So that, although in a few instances the notes were taken by others, the Author holds himself personally responsible for the accuracy with which the cases have been observed, and the fidelity with which they have been reported.

There are certain views of treatment pervading these pages which have resulted from the observation of disease in general, and of facts more numerous than those brought forward in this work. These relate to the substitution of mercury for venesection in acute inflammations, more especially those affecting serous and fibrous membranes; and to the employment of tonic in combination with sedative remedies in a large class of chronic disorders. Such views are not peculiar to the writer; but still they are by no means universally adopted, or carried out in practice. As they are, in this instance, principally founded on cases that have occurred in a populous manufacturing district, they may be considered by some as inapplicable to the inhabitants of other localities, where the nature of the employment and the surrounding atmosphere have a more invigorating effect on the constitution. A slight allowance may possibly be required for this difference in circumstances; at the same time, it must be borne in mind, that many cases were observed among the higher classes, to whom this remark does not apply. Some old practitioners, of sound judgment, are of opinion that depletion can be less borne by persons in general at the present time than it could be forty or fifty years ago. There is probably some truth in this; for, in proportion as civilization advances, and commercial enterprise is extended, the mind is more exercised, and the nervous system more taxed, so that to a certain extent the constitution is thereby impaired and debilitated. Be this as it may, it will hardly be denied by any that venesection has been, and is still practised, with too little caution and discrimination.

The principal object having been, not so much to produce a systematic treatise, as to record the results of personal experience, little reference has been made to the works of previous writers. This, it is hoped, will be excused, and attributed to the nature of the general design and a want of leisure, rather than to any desire on the part of the Author to underrate the labours of others, in every respect more valuable and important than his own.

An attempt has been made, with the aid of one of Powell's best microscopes, to determine the nature of certain morbid products. It is right, however, to state, that these investigations, although conducted with an anxious regard to accuracy, are less satisfactory to the Author's mind than they might have proved had his experience in the use of the instrument been more extended.

The principles of auscultation have been purposely developed in a very elementary manner; for it must have been felt by all those who are engaged in clinical teaching, that one of the greatest difficulties they have to contend with arises from the incomplete preliminary education of some of their pupils, and their imperfect acquaintance with the laws of physical science.

In order to assist the student, and to prevent repetition in referring to the laws of sound, a short summary of such of its properties as are applicable to auscultation of the chest, has been prefixed to the chapters on that subject.

Should any of the opinions which are scattered through the following pages appear to be novel, they will doubtless be received with caution, or they may be altogether rejected. In either case the facts will remain, to furnish materials for the elucidation of truth in abler hands, and possibly to afford assistance in the discovery and treatment of disease.

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PRACTICAL OBSERVATIONS

ON CERTAIN

DISEASES OF THE CHEST.

CHAPTER I.

PROPERTIES OF SOUND.

I. *Sound is perceived when an impulse of a certain force and suddenness is mechanically given to matter in communication with the acoustic nerve.*

Some movements, as the slow waving of a rod through the air, do not give rise to appreciable sound; hence a certain degree of force and suddenness is required, as instanced in the cracking of a whip. Although air is the usual medium through which sound is conveyed to the ear, any solid, liquid, or aeriform matter suffices for this purpose.

II. *When the impulse is single, or when one impulse follows another in very slow, irregular succession, the sound perceived is called* NOISE*; when they reach sixteen in a second* CONTINUED SOUND *is produced; and when they succeed each other at regular intervals and reach thirty-two in the same time, a* MUSICAL NOTE *results.*

Thus if a toothed wheel be very slowly turned, and a steel spring be made to bear against its circumference with a constant pressure, each tooth as it passes will receive an equal blow from the spring, and give rise to a separate *noise.* If the velocity of the wheel be increased so that the spring shall strike more than sixteen teeth in a second, the noises thus engendered will run into each other, and produce a *continued sound;* and if the wheel be turned with a still increased and uniform velocity, so that thirty-two blows shall be given in a second, a *musical sound* or note will result. The blow of a hammer makes a *noise;* the rumbling of a carriage a *continued sound;* and the blast of a horn, or other such instrument, a *musical note.*

III. *The tendency of elastic bodies and their particles to regain the position from which they have been displaced by any impulse, gives rise to a to-and-fro motion or vibration, whereby the primary impulse is repeated, and a continuous sound or musical note is produced by a single blow.*

It is by these vibrations that musical sounds are generally produced; being sometimes excited in flexible bodies made tense, as the cords of a violin, harp, or pianoforte, and the parchment of a drum; sometimes in rigid bodies, as bells, glasses, and the steel bars of musical boxes; at other times in confined columns of air, as in the different wind instruments.

The vocal apparatus is a wind instrument, the mechanism of "which may be considered as consisting of *lungs* or *bellows,* capable of transmitting, by means of the connecting windpipe, a current of air through an apparatus contained in the upper part of the windpipe, which is termed the *larynx.* This apparatus is capable of producing various musical notes which are heard after passing through a *variable cavity,* consisting of the pharynx, mouth, and nose."[1] In the larynx, or mouthpiece, is a narrow slit called the *glottis,* caused by the contraction of the windpipe in its transverse dimensions, the edges of which are formed by white, fibrous, elastic ligaments, called vocal cords. Professor Willis has shown that sound is produced by the vibration of these ligaments when their opposite surfaces are placed parallel one to the other, as in Fig. 1; but that no sound takes place when they are in any other position, as in Fig. 2.

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[1] Professor Willis on the Mechanism of the Larynx, in the Cambridge Phil. Trans. vol. iv, part iii, p. 324.

Fig. 1.

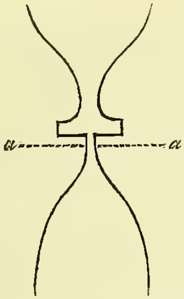
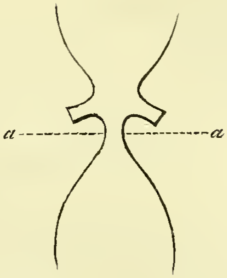


Fig. 2.



a—vocal cords. [From Prof. Willis.

He has also shown that the pitch of the sounds thus produced depends on the degree of tension of the vocal ligaments. A great difference, however, exists between the vocal apparatus and ordinary wind instruments, owing to the manner in which the sounds can be modified by the *variable cavity;* and which cannot be here considered.

Vibration thus taking place in elastic bodies, it follows that—

IV. *Bodies are sonorous in proportion to their molecular elasticity.*

Thus clay, putty, and such like inelastic substances, give out a dull heavy noise on being struck; whilst elastic bodies, steel, glass, &c., &c., give out a musical note. Large masses of matter, however, whether solid, liquid, or aeriform, cannot take on a strong vibration so readily as smaller masses, because the free excursion of their molecules is prevented by the extent of the mass. The most sonorous bodies, therefore, are strings, rods, thin membranes or plates, and small confined columns of air.

V. *Sound is propagated through matter by these vibrations which take the form of waves.*

A notion of the formation and progress of the waves of sound may be gathered from observing the to-and-fro waving in a stretched cord that has been struck, or the waves excited by dropping a stone into the water. It is quite apparent that these waves are independent of the motion of the mass of the cord and of the water.

VI. *The breadth of the waves of sound, or the distance of one from the other, varies inversely with the number of vibrations which occur in a given time.*

If the waves be observed in cords of different weights and lengths, and put to different degrees of tension, it will be seen that the shorter, lighter, and tighter the cord, the less will be the breadth of the waves. Now tension increases the elasticity of the cord, and causes its molecules to return more quickly to their place of rest after having been struck, and thus the number of its vibrations in a given time is increased. So also the diminution of the length and weight of the cord produces a similar effect as in the analogous case of the pendulum. Hence the breadth of the wave diminishes with the increased velocity of the vibrations, and *vice versâ.* So also the breadth of the waves in a confined column of air will depend on its size and specific gravity.

Propagation of sound being thus dependent on a continuance of its vibrations in some matter or other up to the ear, it follows that—

VII. *Matter is a good conductor of sound in proportion to its elasticity and sonoriety.*

Hence the transmission of sound through the atmosphere is affected by its specific gravity. Thus, in consequence of the air becoming more and more rarefied as we ascend in the atmospheric column, sound is badly propagated at the summit of high mountains; and it is, on the other hand, more freely conducted by a condensed atmosphere, such as exists in a diving bell. Heat increases the velocity of sound, while it diminishes its intensity. Now if a vacuum were supposed to exist, there would be no matter to carry on a vibration, and we might therefore expect that sound would not travel at all in vacuo. Such is the fact, as has been proved by suspending a bell in the middle of the chamber of an air-pump. The sound of the bell becomes fainter and fainter as the air is withdrawn from the chamber, and ceases altogether when a vacuum is approached.

VIII. *The waves of sound travel at an average rate of nearly eleven hundred feet per second in air, four times faster through water, and from eight to seventeen times faster in solids according to their molecular construction.*

IX. *The waves of sound diverge in all directions, and hence the intensity of a given sound decays in receding from its origin as the square of the distance increases.*

In this respect they follow the same law as the rays of light and heat.

Some sounds, however, do not spread equally in all directions. Thus if a tuning fork be struck, and then slowly turned round its axis about a foot from the ear, at every quarter of a revolution the sound will be found to increase and decrease alternately. The maximum of sound is heard when the flat surface is either parallel to or at right angles with the outer ear, and its minimum when the flat surface of the fork is at an angle of 45° to it.

There are two ways in which the decay of sound may be lessened. The one by confining its waves, if aerial or fluid, respectively in tubes containing air or water; or if solid, by rods. This will be more properly considered under the head of Reflection. The other way consists in bringing up quickly to the ear the matter in which the sound originates, or through which it is being propagated, before any great divergence can take place. Thus a tuning fork having been struck, may be quickly brought up to the ear. The wind has a remarkable effect in thus increasing or diminishing the intensity of sounds in certain directions, according as it blows to or from the point of hearing. The sound of church bells is often thus carried to a great distance.

X. *The intensity of different sounds heard at equal distances from their origin depends on the force of the primary impulse, and the sonorous nature of the material in which they are excited, and the consequent excursion of the wave.*

The excursion is quite independent of the breadth of the wave. Thus in water the excursion would be measured by the height of the wave above the ordinary level of the water, and its breadth by the distance of the crest of one wave from that of the one next to it. The difference between the two will be clearly recognised in looking at the vibrations of a long cord that has been struck at right angles to its direction. Whether struck strongly or feebly, the breadth of the wave running along the cord will be the same; but the stronger the stroke the greater will be the lateral bulge or height of the wave. But the string thus follows the law of the pendulum; for the further it is driven the faster it returns, so that the number of vibrations in a given time is the same in a strong as in a weak stroke; and consequently the breadth of the wave remains the same.

XI. *The waves of sound on reaching a medium different from that which they are traversing are partially reflected.*

No sounds are totally reflected, because there is no matter, however different in structure from that in which they were first passing, that will not enter into some kind of vibration; and hence some part of the wave is propagated onwards in its original direction through the new medium. The reflection of sounds in the air from smooth solid surfaces is familiar to all, by its occasionally giving rise to echo; and it is a fact of every day observance that a wall, or any other projecting solid, interrupts the progress of aerial sounds, and reflects them back. The same takes place in water; indeed, fluid vibrations, especially when they meet the air obliquely at the surface of the water, are almost totally reflected; this may be verified by striking a bell under water. So, also, if sound be engendered in a solid mass, the ear placed very close to it, but not actually in contact with it, may be unable to detect a trace of it through the intervening air, but will hear it distinctly on being laid on the solid body. Hence, in order to prevent this divergence of sound, and to convey it a long distance with as little decay as possible, a narrow portion of a similar material to that in which the sound is generated is employed; such as tubes of air and water respectively for aerial and fluid, and a rod for solid vibrations. By this means the waves are reflected to and fro from the surrounding medium, till they reach the ear. On this principle speaking and hearing trumpets are constructed.

XII. *The waves of sound are partially refracted when they fall obliquely on a different medium.*

This takes place at a variety of angles, and thus "the general wave is broken up into a number of non-coincident waves, emanating from different origins, and crossing and interfering with each other in all directions. Now whenever this takes place, a mutual destruction of the waves, to a greater or less extent, arises, and the sound is stifled and obstructed."[1]

From these two last properties of sound we may conclude that—

XIII. *Mixed media are bad conductors of sound.*

Such are all porous substances, sponge, linen or woollen cloths, where there is an alternation of solid matter with that which is fluid or aeriform. Also all bodies that have cracks in them, or are imperfectly united to other bodies. The waves of sound in traversing such substances are constantly passing from one medium into another; and as it has been shown that each of such changes is accompanied by reflection and refraction, the sound soon becomes stifled and lost. This has been elegantly illustrated by Chladni. Having produced a musical note from a glass goblet, he poured into it champagne in a state of effervescence, on which the sound ceased, but reappeared immediately on the subsidence of the effervescence. In this case the air within the glass was for a time replaced by a mixed medium, consisting of bubbles. For similar reasons the hygrometric state of the atmosphere affects the transmission of sound through it.

We may also conclude that—

XIV. *Sounds are best conducted by the media in which they are generated.*

This necessarily results from the fact that any change of medium is attended with loss of sound. The sound of voices in a closed room, inaudible to a person without it, is immediately heard when by the opening of the door, the air is allowed freely to unite the voices of the speaker with the ear of the listener.

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[1] Encyclopædia Metropolitana. Mixed Sciences, vol. ii, part 105, p. 776, by Sir J. Herschel.

XV. *The pitch of a musical note ascends from grave to sharp as the number of impulses or vibrations in a given time increases, and consequently as the breadth of the sonorous wave diminishes.*

Musical notes are divided into sets of seven each, often familiarly but incorrectly termed octaves, each having a certain number of impulses in a constant ratio to the other. Thus, if the lowest note in the octave is made up of twenty-four vibrations, or some multiple of it, the scale would run thus:

24	27	30	32	36	40	45	48
1st	2d	3d	4th.	5th	6th	7th	8th

Therefore the eighth note, or octave, has twice as many vibrations as the first; and so each note in the octave has double the number of vibrations of the corresponding note in the octave below it, and half the number of that in the octave above it.

XVI. *When a simple numerical proportion exists between the number of impulses which form two or more notes sounded together, the notes are said to be in harmony with each other.*

The more closely the numbers approach to each other, the more pleasing is the harmony. Such as 1 and 1, 1 and 2, 2 and 3, &c. Five is the highest prime number that enters into harmony. Let us apply this to the scale

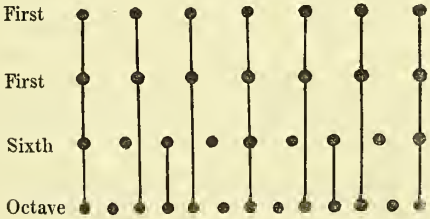
1st and 3d = 24 : 30 = 4 : 5
1st and 4th = 24 : 32 = 3 : 4
1st and 5th = 24 : 36 = 2 : 3
1st and 6th = 24 : 40 = 3 : 5
1st and 8th = 24 : 48 = 1 : 2
8th and 6th = 48 : 40 = 6 : 5
4th and 6th = 32 : 40 = 4 : 5
8th and 4th = 48 : 32 = 3 : 2
7th and 5th = 45 : 36 = 5 : 4
&c. &c.

Thus, the 1st, 3d, 5th, and 8th, may be sounded together in harmony, and the 1st, 4th, 6th, and 8th, and any combination of them. But other combinations, like the following, where the prime number is higher than 5, are called discordant.

1st and 2d = 24 : 27 = 8 : 9
2d and 3d = 27 : 30 = 9 : 10
&c. &c.

Now the simpler the proportion between the number of impulses which constitute the different notes sounded together, the oftener do they coincide or fall together. Thus, by the annexed table it is seen that when two notes of the same pitch

Fig. 3.



are sounded together, every impulse of the one coincides with that of the other. When the first and sixth occur together, every fourth beat of the first coincides with every fifth beat of the sixth. When the first and octave are sounded together, every beat of the first coincides with every other beat of the octave. When the sixth and octave are sounded together, every third beat of the sixth coincides with every fourth beat of the octave; and so forth.

Thus musical notes are in harmony with each other in proportion to the coincidence of their sonorous waves. The jarring unpleasant sound produced by the non-coincidence of the waves in two deep-toned organ pipes out of tune is a sure guide to the tuner when the ear has some difficulty in appreciating the depth of tone.

XVII. *When a musical note is excited in any matter, and another portion of matter in communication with it gives out a similar note by sympathy, this last body is said to consonate with the former.*

Thus one of the strings of a harp or pianoforte may often be heard to repeat the sustained note of a person singing, and thus consonates with the larynx. The explanation of this would require reference to the laws of vibration of different bodies, which cannot be discussed here.

XVIII. *Quality of tone depends on the form of the sonorous wave.*

Every noise and sound that is heard has its characteristic quality of tone, which can be appreciated by the ear with the greatest possible accuracy. Thus sounds differ from each other in *pitch, intensity,* and *quality.* Now the wave by which a mechanical impulse is propagated may be supposed to be subject to alterations in respect to *size, extent of excursion,* and *form.* As, therefore, the pitch and intensity of sound respectively depend on the *size* and *extent of excursion,* the quality of tone may be concluded to depend on the *form* of the wave. This was Euler's opinion.

There are other reasons for this supposition, which would be out of place here.

It is well known that the quality of tone of musical instruments depends in some degree on the material of which they are constructed.

CHAPTER II.

SOUNDS ELICITED BY PERCUSSION.

There are certain sounds which are elicited from the chest by striking it, and others which are produced by the acts of respiration, the exercise of the voice, and the movements of the heart.

On Percussion. The chest, considered in reference to sound, may be compared to a drum, the sonoriety of which depends on the vibration of its parchment, and varies with the weight and tension of that membrane, and of the medium in which it vibrates.

Thus, were the parchment thickened by layers of paper or leather fastened to it, or made very slack, the note of the drum would not only become graver, but its sound would be deadened; and were the cavity of the drum filled with sponge, the same results would follow; and if filled with sand, nothing more than a short dull noise would be given out by it.

The frame of the chest, consisting of thin flat bones fastened together by ligamentous joints in one part, and by elastic cartilage in another, and in ordinary cases covered only by a moderate layer of muscles, fat, and skin, is favorably constructed for resonance on being struck. Its cavity is not filled with mere air, like the drum, but with a spongy elastic substance, the lungs, containing within their cells a great quantity of air, and therefore allowing of very free motion of its walls.

The chest therefore on being struck gives out a hollow sound, very different from that which is elicited in a similar manner from the arms or thighs. Hence anything which tends to increase the elasticity of the frame, or to lessen its weight, or that of the contents of its cavity, will increase its sonoriety; whilst a contrary effect will be produced by an addition to their weight, and a diminution of their elasticity.

We must therefore inquire in what manner such modifying circumstances arise both in *health* and in *disease.*

Percussion of the Thorax in health.

The elasticity of the frame of the chest may be directly affected by age, inasmuch as the cartilages of the ribs become gradually ossified as old age approaches, and thus the elasticity of the thoracic skeleton is diminished. In many cases this is counterbalanced by a decrease of fat, whereby the weight lying on it is lessened, and by an increase in the size of the air-vesicles.

So again a great difference will exist in the sonoriety of the chest in fat and lean persons of the same age. All parts of the chest are not covered alike with muscles and fat, and in one spot the scapula also lies on the ribs. Those parts therefore which are most thickly covered, will sound duller than those which have less weight on them. Thus the margins of the middle portion of the sternum and the axilla being least coated, will give out the clearest, the region of the scapula and the pectoralis muscle the dullest sound.

In persons who work heavily with the right arm, the right pectoralis muscle is often so much more developed than the left, that the sound is very sensibly duller over the right muscle than over the corresponding one on the left side.

In respect to the contents of the thorax, if the spongy lung should be in any part replaced by air, percussion would elicit a clearer sound over that spot than over other parts; and if it should be replaced by solid or fluid matter, an opposite effect would be produced.

When the chest is in a healthy state, the lungs are not displaced by air unless in one particular case, and that is when the stomach, being distended by gas, rises up into the left side of the chest above its natural level. The sound to which this gives rise is of a peculiar ringing character, which a practised ear can at once recognise. On the other hand, the stomach may be unusually distended with liquid, and then a contrary effect results.

Besides the lungs, however, the frame of the chest contains the heart and large vessels. The heart touches the surface of the chest, and is uncovered by lung, over a space varying from the size of a shilling to that of a half-crown piece, in health. Over this spot therefore the free vibrations of the walls of the chest are opposed, and *consequently* a dull sound is given out on percussion. The liver occasionally rises higher up into the right side of the thorax than usual, a condition not unfrequently induced in females by tight lacing; and in that case the lungs are replaced by so much solid matter, and dullness on percussion is the result.

Percussion of the Thorax in disease.

The weight of the frame of the chest may be increased in particular spots by the presence of tumours, whether consisting of solid matter or containing fluid; and thereby its sonoriety may be diminished. The same effect may be produced by the effusion of serum into the cellular tissue, this being usually more diffused than the former.

A great many diseases tend to alter the character of the contents of the thorax, some diminishing their density, and others increasing it. Of the former kind are pneumo-thorax, or that in which air exists between the two surfaces of the pleura, and thus supplants the lung; and emphysema of the lungs, in which the air-cells are dilated, and in some cases ruptured, whereby the air in such parts of the lung bears a much greater ratio to the solid walls and vessels than it does in a healthy state; and also some large cavities in the lung generally of a tubercular character, which, when containing but little fluid, are necessarily filled with air. All these diseases produce the same effect in respect to percussion of the chest, rendering the sound much clearer than it is in health on the principle above stated.

The density of the contents of the thorax is increased by the presence of fluid in the pleural cavities. In some instances the fluid is limited by adhesive bands, and the resulting dullness may be under such circumstances confined to one spot of the chest, but more usually it is bounded only by the pleural sac. In that case, if in large quantity, the whole side will sound dull on percussion; but if less fluid exist, the seat of dullness will vary with the position of the body, and will follow the fluid, which will gravitate to the lowest parts.

When there is a communication between a pleural cavity which thus contains fluid, and one of the bronchial tubes, as in most cases of pneumo-thorax, then the superior part of the chest containing air, and the lower part fluid, the former sounds clearer, and the latter much duller than is natural.

So also the pericardium may be distended with blood, serum, or pus. Collections of fluid may also occur within the organs of the thorax. Thus the heart may be dilated and contain consequently an unusually large quantity of blood, whereby the extent of dullness in the precordial region may be increased.

The aorta and pulmonary artery may be so dilated as to reach the surface of the chest, or an aneurism may be formed by the rupture of their coats, which may produce the same effect.

In regard to fluid in the lungs, as long as it is confined to the bronchial tubes, the sound of the chest on percussion is not affected, because there exists an abundance of healthy and elastic lung between the tubes and the surface of the chest. Dullness, however, will result from serous or sanguineous congestion of the cellular portion of the lungs in contact with the walls of the thorax, whether of a passive character, being the result of obstruction to the circulation; or of an active character, as in the early stages of pneumonia; and also from abscesses or excavations containing fluid and approaching the surface.

Solid matter may also replace the lung and give rise to dullness. The heart may be hypertrophied, and there may be strumous or encephaloid disease in the mediastinum coming up to the surface. Tubercles or gray granulations may be thickly clustered in the lungs; pneumonia, chronic or acute, may exist; and coagulated blood may be found, as in pulmonary apoplexy; and also encephaloid disease, both in a hard and softened state. In all these cases the chest will sound dull over the spots corresponding to these solid deposits. It must be remembered, however, that a considerable quantity of solid matter may exist in the centre of the lungs, and also may be diffused in small masses among healthy lung, as in some cases of acute phthisis, without engendering dullness of sound.

A sound has sometimes been elicited by percussion, which has been styled the *bruit de pot fêlé*, from its similarity to that which is produced by striking a cracked pitcher. It has been conjectured to arise from the forcible expulsion of a quantity of air from a tubercular excavation into the air-tubes leading to it. If this were the true explanation, we should not find it appearing and disappearing in the same individual, which may not unfrequently be observed. This fugitive character, as well as the nature of the sound itself, renders it much more probable that it arises from the splash of fluid and air mixed, and that a certain proportion between them is necessary for its production, a proportion that is ever varying.

CHAPTER III.

AUSCULTATION OF THE SOUNDS OF RESPIRATION.

When the ear is placed in contact with the walls of the chest of a person in health, certain sounds are heard during respiration, and others distinct from and independent of that act.

Sounds of Respiration.

During the whole of the time the air is entering the chest, a soft sound is heard by the ear in contact with its surface, which has been accurately likened to that which is produced by a person sipping the air with his lips. Sometimes this sound is prolonged so as to accompany the escape of the air from the chest; always, however, in healthy persons being less intense and less prolonged during expiration than during inspiration; according to some writers bearing a ratio in these respects of one to five. Practical auscultators, however, will I think readily admit that in the great majority of persons no sound, or at most only a short puff, is heard during expiration. This sound has obtained the name of *pulmonary* and *vesicular respiration* or *sound.*

At certain parts of the chest, more especially between the shoulder-blades, the sound during inspiration is often coarser than at other parts, and that attendant on inspiration is both more intense and more prolonged. This has been called *bronchial respiration* or *sound.*

Over the trachea the sounds during inspiration and expiration are found to be nearly if not quite equal in duration and intensity; and both are much stronger than that heard over the surface of the chest, and possess a peculiar, hollow, blowing character, as if wind were blown up a tube into the ear. This has been named *tracheal respiration* or *sound.*

These sounds are occasionally modified in respect of their *intensity* and *quality,* by circumstances which occur both in *health* and *disease.*

We have then to inquire, in the first place, how these sounds are *formed,* and in the next place how they are *modified.*

Formation of the natural Respiratory Sound.

There are certain movements which take place during respiration, and which may consequently give rise to sound. These are:

1. The movement of the frame of the thorax, which turning on its hinges at the spine, rises and falls during respiration.
2. The contraction of the muscles by which this movement is effected.
3. The gliding of the lungs on the ribs during their expansion and retraction.
4. Movements within the structure of the lungs.
5. The movement of the air rushing in and out of the lungs, and impinging on the air-passages, from the mouth to their vesicular terminations.

All these movements may possibly produce sound, but the question to be solved is, which of them produce such an amount of sound as to be appreciable to the ear, placed on the chest and trachea.

The sounds of the to-and-fro movement of the frame of the thorax must take place both during inspiration and expiration, and therefore cannot form the pulmonary sound, which is in most cases almost inaudible during the latter act: *a fortiori,* it cannot be propagated towards the larynx and form the louder tracheal sound.

The same reasoning applies to the sound which might arise from the lungs rubbing against the ribs, and from inter-pulmonic movements, supposing it possible that audible sound could be thus engendered.

The sounds produced by the friction of the inspiratory muscles during their contraction, are formed only during inspiration, and so far fulfil the condition necessary for the formation of the pulmonary sound. But the contraction sound of muscles, much more powerful than those which expand the chest, is much less intense than the respiratory sound; and, moreover, if the ear is placed on the chest during a forced expiration, no additional sound is detected. This movement must, therefore, be excluded from giving rise to appreciable sound.

There remains, therefore, only the last-named movement as the cause of the respiratory sound; viz. that produced by the air entering and leaving the lungs, and impinging on the air-passages.

We have now to inquire how the *tracheal, bronchial,* and *pulmonary* sounds are thus produced by the friction of the air, each having their peculiar character.

Tracheal Sound.

It is evident that the coarse, hollow, *tracheal sound,* heard alike when the air enters and leaves the lungs, is principally formed in the trachea and larynx, the part of the air-passages which is traversed by the whole of the air required for respiration, and is strengthened by the sounds which are formed in the mouth on the one hand, and in the bronchial tubes on the other, and which are propagated up to that point in the trachea over which the ear is placed.

Pulmonary Sound.

A variety of opinions have been held concerning the seat of the formation of the pulmonary sound. Some maintain that it is formed in the mouth, fauces, or glottis, and is thence propagated to the surface of the chest; others, that it is formed in the air-vesicles; and others in the middle-sized and small bronchial tubes.

In endeavouring to determine this point, it must be constantly borne in mind that the great characteristic of the pulmonary sound is its marked diminution or entire absence during expiration; and that it cannot be produced by any cause which does not satisfy this condition.

Laennec called this sound vesicular, because he supposed it to be produced by the air entering and leaving the vesicular terminations of the air-tubes; but he has not attempted to account for its absence or diminution during expiration. Dr. Skoda, however, endeavours to account for this difference, on the supposition that a greater resistance is offered to the entrance of the air into the vesicles than to its exit from them. This, he argues, is effected by the resilience of the vesicles, which to a certain degree resists distension. This would be true if the air were forced into the trachea, whilst the walls of the chest were passive, but not otherwise. In fact; however, the air-vesicles are similarly situated to a bladder placed under the bell of an air-pump, and communicating with the external atmosphere through its neck. If this communication be for a moment interrupted, by turning a stopcock which has been fixed in the neck of the bladder, and the pump be worked, then as the air around the bladder becomes rarefied, that within it becomes proportionately rarefied, expands, and exerts an equable outward pressure against the internal surface of the bladder, with little or no friction. The stopcock being then turned, the external air rushes in. This is just what takes place during inspiration, under the influence of the muscles which elevate the ribs, and enlarge the capacity of the thorax; except that there is no appreciable interval between the expansion of the air already in the vesicles and the entrance of additional air from the bronchial tubes. No greater resistance, therefore, is offered to the entrance of the air into the vesicles during respiration than to its exit from them, and consequently no more sound can be engendered in them during inspiration than during expiration. Hence it follows that the respiratory sound, which is more intense and prolonged during inspiration than during expiration, is not principally formed in the air-vesicles.

M. Beau, Dr. Spital, and others are of opinion that the pulmonary sound is chiefly formed in the fauces and glottis. In support of this opinion it is asserted that if the glottic aperture be enlarged by a peculiar movement of the muscles of the larynx, the resistance to the air at this spot will be thus removed, and it will enter the lungs without giving rise to sound. In order to prove that the lungs are equally inflated in the same given time, when the sound is thus prevented, as when it is produced by ordinary respiration, a person is made to respire from a pig's bladder filled with air. A slight inspection might probably convey the impression that such was the case; but if the bladder be placed in a glass vessel with a long narrow neck containing coloured water, and if the rise and fall of the water be accurately observed, and the time consumed by each respiration noted by means of a stop-watch, it will be found that when the respiratory sound at the trachea is very much weakened, less air enters the lungs in a given time. If, therefore, the force with which the air enters the lungs is thus diminished, the respiratory sound at the surface of the chest, never very strong, will of course cease, whatever be the seat of its generation. It is further asserted that the intensity of the respiratory sound at the surface of the chest is increased by stertorous noises produced in the mouth and fauces. The pulmonary sound may certainly be drowned by this means, and also by base-viol sounds if they are sufficiently loud; but it is not strengthened. On the contrary, when such noises are not very loud, they are perceived to be quite distinct from the pulmonary sound, which remains unmodified by them. Careful and unbiassed observers, well accustomed to auscultation, will, I think, have no difficulty in deciding on the comparative correctness of these opposite statements.

M. Beau endeavours to account for the difference between the intensity of the pulmonary sound during inspiration and expiration, by assuming that in the former act the glottis contracts, and offers an obstacle to the entrance of the air which is not offered to its exit. In that case the difference in the intensity of the two sounds should be quite as much marked over the larynx as at the surface of the chest; whereas, in fact, there is little or no difference perceptible in the former spot. If the pulmonary sound were formed in the mouth and larynx, and thence transmitted to the surface of the chest, *a fortiori* the stronger sound of the voice would be conveyed there also. It will presently, however, be seen that in health no resonance of the voice is perceived over the greater part of the chest. It would appear, therefore, that the sounds engendered in the mouth and trachea are so weakened by divergence, and by destruction in the non-homogeneous tissue of the lungs, that they do not form a principal part of the pulmonary sound. If, then, the pulmonary sound, which reaches the ear over the surface of the chest, is not engendered to any great amount at either extremity of the bronchial tree, it must be chiefly formed in the intermediate parts, the bronchial tubes.

It must, however, be either originally formed louder, or be more freely propagated to the ear during inspiration than expiration.

In a very ingenious Paper, read by Dr. James Carson, of

Liverpool, in 1841, at the meeting of the British Association for the Advancement of Science, at Manchester, it is stated to be the opinion of the writer that the muscular fibres, which surround the smaller bronchial tubes, contract during inspiration for the purpose of narrowing the tubes and thereby forcing the air up into their vesicular terminations. If such be the case then an obstruction is offered to the entrance of the air which exists only at the very commencement of its departure from the lungs, and ceases on the relaxation of the muscular fibres.

Before I was made acquainted with these views, I was unable to account for the inequality of the two sounds on any other grounds than the difference in the circumstances which affected their propagation to the ear; considering that as, during inspiration, the current of air sets towards the surface of the chest, it would quickly carry up the sound, and thereby, to a certain extent, diminish its absorption by the mucous membrane of the tubes, which might take place to a greater extent during expiration when the current sets in the opposite direction. But this does not apply to that portion of the sound which is propagated by the sides of the tubes. But, even were this not so, I am now perfectly satisfied that any such cause is insufficient to produce the great difference in the intensity and duration of the two sounds. For if a person respire through a metal or smooth wooden tube, the two sounds which are heard at the other end of the tube, during inspiration and expiration, will be found to be of equal intensity; but if the same person respire through a tube made of a roll of carpet, the sound produced when the current of air sets towards the ear, will be found to be rather louder than that which is found when the air is drawn from the ear. This difference, however, is so slight that it is impossible that that which is observable between the two sounds, when heard at the surface of the chest, can be attributed to this cause alone.

It may be concluded, therefore, that the pulmonary sound is principally formed by the air rushing through the smaller bronchial tubes.

Bronchial Respiration.

The reasons why bronchial respiration is heard between the scapulæ can now be understood. In this spot the large bronchial vessels approach the surface of the chest, and consequently the coarse to-and-fro sound, which is formed by the air entering and leaving the lungs, is plainly heard. But as a certain portion of lung intervenes between the tubes and the ear, a certain amount of pulmonary sound is joined with the bronchial sound during inspiration, and makes it fuller and slightly more intense, than that which is heard during expiration.

We have now to consider the modifications which, under certain circumstances, are produced in the *intensity* and *quality* of the respiratory sounds.

Sometimes the intensity of the pulmonary sound is *increased* above its normal standard over particular parts of the chest, at other times it is *diminished*, or disappears altogether; it is also frequently *modified* by the mixture of other sounds with it, or *replaced* by the bronchial or laryngeal sound.

The *intensity* with which the pulmonary sound reaches the ear at the surface of the chest may depend on circumstances which affect its *formation* or its *propagation.*

Age has a great effect on its formation. In youth the frame of the chest, fastened together by cartilage, is very elastic, and easily expands. Accordingly during this period of life respiration is carried on very vigorously. A considerable quantity of air rapidly enters the chest; in consequence of which the intensity of the pulmonary sound is great, and has obtained the designation of puerile respiration. With the progress of age, however, the costal cartilages become replaced by bone, and thus the elasticity of the frame of the chest is diminished; the walls of the air-cells also gradually become less and less resilient, and the act of respiration becomes slower and less vigorous. All these circumstances combine to weaken the pulmonary sound as old age creeps on.

When the whole or a large portion of one lung is rendered impervious to air by disease, the intensity of the pulmonary sound is often found to be greatly increased over the healthy lung. This arises from increased energy of the act of respiration, by which means the impermeability of the diseased portion of lung is to a certain extent compensated for.

When the costal and diaphragmatic pleura is inflamed and painful, the chest is less freely expanded, and in this manner the force of the respiratory act is diminished, and the intensity of the pulmonary sound lessened.

The elasticity of the parenchyma is sometimes affected by chronic bronchitis, and pulmonary emphysema. In exaggerated cases of the latter disease the respiratory current is so much diminished in some spots that the pulmonary sound totally ceases.

The lungs may also be prevented from expanding by the pressure of fluid in the pleural cavities, and the sound thus lessened or destroyed.

The air may be prevented entering certain of the bronchial tubes by the external pressure of a tumour, or the deposit of solid matter within them or their vesicular terminations. External pressure may be caused by diseased bronchial glands, or aneurism of the aorta. (Cases IX, X.) The large tubes may be blocked up by solid matter in encephaloid disease of the lung. (Case LXXXIX.) The middle-sized tubes are not unfrequently plugged up by pellets of tough mucus; but in general the absence of sound thus caused is only temporary, and it is restored by a cough or a strong respiration.

But the most frequent cause of the suspension of the respiratory sound is the deposit of solid or liquid matter in the air-vesicles and minute bronchial tubes, whereby their expansion is prevented, and with it the passage of the air through those tubes in which the pulmonary sound is principally engendered. This occurs in phthisis, pneumonia, cancer, and pulmonary apoplexy.

If it be allowed that a great part of the pulmonary sound results from the obstacles offered to the entrance of the air by the contraction of the circular fibres of the small bronchial tubes, then if their action should be impaired, the pulmonary sound would be at the same time destroyed.

In certain cases the pulmonary sound is jerking, having two or three interruptions during each inspiration. I have not been able to satisfy myself of the exact condition on which this depends, having observed it both in incipient phthisis, and also in persons who had no symptoms of this disease.

The intensity of the pulmonary sound may also be affected by its *propagation*. This must take place through the walls of the chest, which will transmit the sound from within with greater or less intensity according as they are thin, or loaded with fat, serum in their cellular tissue, lymph on their pleural surface, or tumours of various kinds. The pulmonary sound will also be ill-conducted and deadened, if the lung be separated from the sides of the chest by fluid of any kind in the pleural cavities. In this latter case the sound will be impaired both in its formation and propagation.

The pulmonary sound may be *modified* by the mixture of other sounds with it.

It is possible that in certain cases usually called spasmodic asthma, the muscular fibres of the air-tubes are spasmodically contracted both during expiration and inspiration, in which case the same resistance is offered to the exit of the air as to its entrance; and consequently the expiratory sound becomes as loud as that during inspiration. In fact a prolonged expiratory sound is often heard during the paroxysm, which disappears on the breathing becoming tranquil.

In other cases the blood-vessels of the air-tubes may become temporarily engorged, and thus an additional resistance is offered both to the inspiratory and expiratory current, and a coarse double sound produced. In chronic bronchitis the same effect may be permanently induced, and hence the characteristic sound of this disease—coarse, pulmonary sound, prolonged during expiration.

The air-tubes may be obstructed and narrowed at particular spots, and be thus converted into musical instruments, in which the air is thrown into musical vibration by the obstacle offered at the narrow slit. A musical note is thus produced, which being generally more powerful than the pulmonary sound, more or less completely drowns it. As the pitch of such a note depends on the size of the column of air in vibration, which in its turn depends on the size of the tube, a grave note is produced in the large and a more acute note in the small tubes. The former is called the *sonorous rhonchus*, or *bass-viol sound*, from its resemblance to the note given out by the strings of a violoncello, and the latter is styled the *sibilous rhonchus* or *cooing sound*, from its resemblance to the cooing of doves or chirping of young birds in a nest. Not unfrequently these sounds disappear after a cough or deep inspiration, when the obstruction which gave rise to them consists, in all probability, of a pellet of tough mucus.

The obstruction may be insufficient to produce a decided note, but may still impress a musical character on the respiratory sound. It is in this way, probably, that a tubal sound is sometimes formed, particularly if the tube which is obstructed is surrounded by dense solidified lung. In some cases the sound resembles that produced by blowing through a metal tube, and in others almost amounts to the sound called metallic tinkling presently to be described.

When the bronchial tubes are either dilated or open into a cavity, as in phthisis, the respiratory sound is reverberated and strengthened by echo, particularly if the walls of the cavity are hard and lie near the surface of the chest; at the same time the bronchial and tracheal sounds become freely propagated by the air in this tube, and are mixed with and generally drown the pulmonary sound, if it has not already been destroyed by tubercular deposit or chronic pneumonia around the cavity. This sound has a peculiarly hollow character, and has been named *cavernous* or *amphoric respiration.*

In bronchitis, in the first and second stages of pneumonia, and in certain forms and stages of pulmonary congestion, the air-passages contain fluid of various degrees of consistence. In passing through this fluid the air forms with it bubbles, which burst, and thus give rise to moist rattles, the sounds of which vary with the size of the tubes in which they are formed. They may be exactly imitated by introducing gum-water into bent glass tubes of different sizes, and then blowing through them either with the mouth, or by the aid of an Indian rubber bottle affixed to one end of the tubes. If one of these tubes has a bulbous enlargement, like that of an hydrometer, a ringing gurgling is produced in it, by the formation and bursting of very large bubbles, and the echo of their sound in the cavity. This aptly represents a tubercular cavity or bronchial dilatation which gives rise to a similar sound, and which is called *gurgling.* In such cases the sound varies with the quantity of fluid in the cavity, and the density of its walls. Thus, if it be either empty, or quite full of fluid, no bubbles will be formed, from the absence of fluid in the one case, and of air in the other. When the cavity contains a moderate quantity of fluid, there exists the material for the formation of the bubbles and space in which they may burst and resound. So again, if the sides of the cavity are soft and surrounded by spongy lung, there will be but little echo. But if the lung is extensively solidified around it, it will not expand during inspiration, and hence there will be but a slender current of air in and out of it, and therefore not much gurgling will be formed. If now the patient be made to cough, a *splash* will be heard in the cavity during the expiratory effort, produced by the sudden compression of the cavity and the shaking together of the fluid and air. A gurgling will also be heard during the succeeding inspiration, when the air will re-enter the cavity with a force proportioned to that with which it was expelled from it. Accordingly, in lungs which contain tubercular cavities, this noise varies from the gurgling of a large cavity, favorably disposed for its production, down to the click which is produced by the bursting of a solitary bubble in a very minute cavity.

If tubes are employed, from the size of the little finger, down to that of a crowquill, a loud and somewhat unequal moist rattle is heard, which has been termed *mucous rattle*, both when the air enters the tube and when it is withdrawn from it. The larger the tube, the louder is the rattle, and the more ringing is its quality. Now in bronchitis an unusual quantity of mucus is secreted, and in great debility preceding death, a great quantity often accumulates in the air-passages from inability to expectorate. In both these cases, therefore, the mucous rattle is heard, being called the *tracheal rattle* when very loud and ringing; and very properly so, as it is then produced in the trachea. In some cases of chronic bronchitis, the mucus is thick and adhesive; and then we have a succession of large crackings, as if large bubbles were slowly formed and burst; and this is the more marked when the current of air is feeble, as in emphysema. This is probably what Laennec intended to describe by the *craquement à grosses bulles.*

If tubes are made use of, of much less size than that of a crowquill, it will be seen that the bubbles do not burst till they reach the extremity of the tube during the time the air is forced into it from the mouth or Indian rubber bottle. The sound formed by the bursting of these small bubbles at the extremity of the tubes, gives the ear no idea of moisture, but seems like a fine dry crackling. The exact sound is heard in the first stage of pneumonia, œdema of the lung, and in certain forms of pulmonary apoplexy; and here, as in the tube, it is confined to inspiration. It is called the *crepitant rattle.*

The experiment with the tubes would seem to offer a simple and rational explanation of the manner in which this sound is formed in the lungs, and it is the one generally received. In the diseases above mentioned, the fluid is for the most part confined to the smallest sized bronchial tubes and their vesicular terminations. The air rushing through these small tubes cannot form bubbles of a sufficient size to burst, until an increased space is allowed by the expansion of the vesicles in inspiration: in expiration they again contract, and no bubbles burst, and no crepitation is formed.

Dr. C. B. Williams thinks this rattle is produced by the air passing through the fine bronchial tubes compressed by interstitial congestion. In œdema, however, the copious expectoration of serum proves that much of this fluid is contained in the bronchial tubes; and the same occurs in pneumonia, except that the fluid is more viscous and tenacious. But if very thick gum-water be used in the glass tubes, the crepitation will still be produced.

Dr. Walshe attributes the crepitation to the unfolding of the vesicles, the sides of which, he supposes, stick together during expiration. This, however, could only occur at a time when the first stage of pneumonia was running into the second stage, and the vesicles had been nearly obliterated; for in ordinary cases, after expiration has taken place, a sufficient quantity of air must remain in the vesicles to prevent the approach of their sides to each other. But allowing the possibility of the air-cells thus sticking together, they would be separated from each other at the commencement of inspiration, and could not give rise to the crepitation, which is usually prolonged during the whole of that act, and in some cases only appears towards its conclusion. It cannot be argued that some cells are expanded at a later period than others, as is seen when air is forced into the lungs, after they have been removed from the body, because this is not a parallel case; for in inspiration the air is not forced in whilst the lungs remain passive, but enters in consequence of the expansion of the chest, and of the vesicles, which must commence in all of them at the moment the muscles of inspiration begin to act.

When, however, the exact sound can be produced in the manner above stated at the mouth of glass tubes containing viscous fluid, it is reasonable to conclude that its formation in the lungs takes place in a similar manner. That the crepitation in œdema and pulmonary apoplexy, should be less even and dry than in pneumonia, only proves that the fluid extends further into the air-tubes—a fact which is rendered probable by the nature of these affections. In this form it has obtained the name of *sub-crepitant* or *muco-crepitant rattle;* and is frequently met with in the engorgement of the lungs which results from obstruction to the circulation at the left side of the heart, being still more moist and uneven than in œdema and pulmonary apoplexy.

The distinction between a fine or deep-seated mucous rattle, and the sub-crepitant rattle, will be considered by practical men as one of those useless refinements which serve only to confuse the student, and which lead to no practical advantage.

The crepitant rattle, therefore, is considered by Andral and many others, as a fine modification of the mucous rattle, and is most probably produced by the bursting of small bubbles in the air-vesicles.

A sound is heard in certain cases which has been named *metallic tinkling*, from its resemblance to the sound of a very small bell. A similar sound can be produced in two ways: by dropping fluid into a glass decanter partially filled, and also by causing a bubble to burst at the extremity of a moderate sized glass tube plunged into an empty decanter. There is a similar state of things in pneumothorax, when the cavity of the pleura communicates with an air-tube. If the pleura contain both air and fluid, metallic tinkling may often be made to appear by shaking the patient, when some fluid drops into that which is at the bottom of the cavity. When it contains no fluid, the sound may still be heard accompanying respiration, and in that case it may be supposed to arise from a bubble formed at the extremity of the tube which opens into the cavity, and which bursts and reverberates in it. The same sound may be formed in a large tubercular cavity, or a large tube surrounded by solidified lung.

The respiratory sounds may be further modified by being mixed with other sounds engendered *without* the lungs, but still connected with the act of respiration. Thus, although in health, the smooth, lubricated surfaces of the pleuræ play upon each other in a noiseless manner, yet should one or both become coated with lymph, a coarse rubbing sound may be expected. Accordingly such a sound is heard in certain cases of pleurisy. It is called the *rubbing* or *to-and-fro* sound of the pleura, being formed both during inspiration and expiration. When two opposite portions of lymph begin to adhere the one to the other, either closely or by means of bands, the rubbing sound becomes altered in quality, and is turned into a *creaking* sound, something like that of new shoes. In some few instances these pleuritic sounds can be heard at a considerable distance from the body. Some writers imagine that a further modification occasionally takes place by these sounds being converted into a muco-crepitant rattle, on the adhesion becoming more intimate between the two pleural surfaces. It is certainly true that the muco-crepitant rattle is often heard in connexion with pleurisy; it will be found, however, on reference to the chapter on this subject, that there is no constant connexion between this rattle and the rubbing and creaking sounds. Thus it is not uncommon to hear a muco-crepitant rattle during the disappearance of pleuritic effusion, when no rubbing or creaking sound has been heard from first to last; and again, the former sound is sometimes heard at one part of the chest and the latter at another part. More than this, in some cases the muco-crepitant rattle is heard towards the bottom of the back when the patient leans forwards, and disappears on his resuming the upright position, in consequence of effused fluid intervening. In this last case it is clear there is no adhesion to cause this sound; if, therefore, it were pleuritic, it would be the rubbing sound. It is much more probable that the muco-crepitant rattle arises from œdema of the lung—a state which we infer from the frequent occurrence of œdema of the cellular tissue covering the affected side, and also from the copious watery expectoration which so often takes place. On the pleuritic effusion subsiding, the lung begins to expand, and the air is thus allowed to enter it, and meeting with fluid in the bronchial tubes, the muco-crepitant rattle is produced. Accordingly it is at this period of the disease that it is met with.

It now only remains to explain under what circumstances the pulmonary sound is *replaced* by the laryngeal and bronchial sounds.

When the air passes through a bronchial tube, sonorous vibrations are excited, both in the air itself and in the sides of the tube on which it impinges. Hence, if between a bronchial tube, through which the air is passing and repassing, and the surface of the chest, the lung be replaced either by aeriform or solid matter, these vibrations will be freely conveyed up to this spot. Fluid, or any other homogeneous substance, would convey them better than the spongy lung; but by no means so perfectly as air or solids, the former of which would principally convey the vibrations of the air within the tube, and the latter those of its walls.[1] A tube, therefore, with hard solid sides would be the best conductor, as by its means both solid and aeriform vibrations would be conveyed. Next to this, a solid rod of moderate size, passing from the sides of such bronchial tube to the surface of the chest, would best answer the purpose; because the solid vibrations would be reflected to and fro up to the ear in a similar manner to those of air in a tube. A large mass either of aeriform or solid matter would not convey the sounds well, because they would be much weakened by divergence through the mass.

It is on this principle that the bronchial and tracheal sounds replace the pulmonary sound. Thus, when a very large tubercular cavity or bronchial dilatation exists near the surface of the chest, and communicates freely with the trachea by a large bronchial tube, the tracheal sound is propagated up to the ear, and replaces the pulmonary sound, which has been destroyed by obliteration of the smaller tubes. Being thus strengthened by reverberation, a hollow cavernous sound is produced both during expiration and inspiration. Frequently, however, when a large mass of air intervenes, as in some cases of pneumothorax, no respiratory sound is perceived, or at least it is very feeble and distant.

When solidification of the lungs takes place in phthisis, pneumonia, congestion, &c. &c.; the bronchial sound is often conveyed to the ear, with an intensity depending on the degree of solidification, its extent, and situation. Thus, in solidification of the second stage of pneumonia, when the diseased part contains but little fluid, the transmission of the bronchial sound is more complete than in the first and third stages, when solid and fluid matter form a mixed medium. So, again, a whole lung is frequently solidified, and no bronchial sound is heard over any part of it, or else it is very feeble and distant. In other cases a moderate-sized mass of solidification may exist at the surface or centre of a lung, but being separated from the large bronchial tube by spongy lung, very little bronchial sound is propagated by it.

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[1] Prop. xiv.

Laennec, and many of his followers, give a very different explanation of the manner in which the pulmonary sound is replaced over a solidified portion of lung by the bronchial sound. He states that the air, having ceased to enter the vesicles and minute tubes which are blocked up by solid deposit, stops short of them, and passes in and out of the large bronchial tubes alone, and thus only the sound engendered in these tubes is heard. But as the current of air through the bronchial tubes almost entirely depends on the expansion of the vesicles to which they lead, such current must in a great measure cease with the inability of the air-vesicles to expand. Surrounded by cartilaginous rings, we cannot conceive the large tubes capable of sufficient expansion to give rise to any considerable current. The bronchial sound which is heard at the surface of the chest, is not, therefore, formed in the large tubes leading to the diseased portion of lung, but *in those which lead to healthy expansible lung.*

Dr. Jackson[1] supposes that the bronchial sound is drowned by the pulmonary sound in health, but becomes audible when the latter ceases from solidification of the air-vessels. But the expiratory portion of it could not have been drowned, for no sound is heard during the greater part of the time occupied by expiration in health. The bronchial sounds are not drowned in health, but are stifled and destroyed by the intervention of the spongy lung.

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[1] Life of Dr. Jackson, p. 129.

CHAPTER IV.

AUSCULTATION OF THE SOUNDS OF THE VOICE.

When a stethoscope is placed on the trachea of a person in the act of speaking, and the ear is applied to it, the voice seems to mount up the instrument, as if the mouth of the speaker were as close as possible to the ear of the listener. This resonance of the voice is called *laryngophony* or *pectoriloquy.*

When the stethoscope is placed between the shoulder-blades of some persons, the voice seems to issue from the spot on which the instrument rests; this is called *bronchophony.*

Over other parts of the chest no resonance of the voice is perceived in health.

The quality of tone of the voice, both in pectoriloquy and bronchophony, is also more coarse and buzzing than when it is heard issuing from the mouth.

In certain diseases, however, both pectoriloquy and bronchophony are heard at different parts of the surface of the chest, and in some cases their quality of tone is peculiarly modified.

As the formation of the voice in the larynx is unaffected by the position of the stethoscope, or by the diseases in which its resonance is increased over the chest, it follows that the causes of the modifications above mentioned, must be sought for solely in the condition of the parts through which the sounds of the voice pass, and in the influence which it exerts on their propagation to the ear.

When the voice is formed, the vibrations of the vocal cords are communicated both to the walls of the trachea and to the column of air within it; and by both are propagated towards the surface of the chest. No sooner, however, has the trachea entered the chest, than it is divided and subdivided into numerous branches, which become surrounded by spongy non-homogeneous lung, so that the sonorous vibrations of the voice become not only diffused, but broken up and stifled to such an extent, that they do not reach the surface of the chest with sufficient intensity to be appreciable by the ear. Consequently no resonance of the voice is detected.

On either side of the spine, opposite the root of the lungs, however, the larger bronchial tubes approach the surface of the chest; and hence a certain amount of vocal resonance, which constitutes bronchophony, is often heard between the shoulder-blades.

But when the ear is placed directly on the larynx, or on the trachea near it, the divergence which takes place in the air between the voice of the speaker and listener in ordinary conversation is prevented; and the voice is heard in its full intensity. If a solid rod or a stethoscope is interposed, the same effect is produced by the reflection of the sounds up to the ear.

Now as both the sides of the bronchial tubes and the air within them vibrate during speaking, if any portion of the spongy lung which connects them with the surface of the chest be replaced either by aeriform or solid matter, their vibrations will be freely propagated by such matter. Fluid will propagate the sounds better than the non-homogeneous lung, but by no means so freely as air and solids, which are the media in which they were originally engendered. A tube communicating with the trachea will convey the sounds most freely, because both the sides and air within them will respectively propagate the solid and aerial vibrations.

Much must depend on the bulk and situation of these altered portions of lung, as has been fully explained in the remarks made on the propagation of the respiratory sound. Accordingly, when the lung is solidified, bronchophony is often heard, varying with the degree of solidification, and its amount and situation; and when cavities of moderate size, surrounded by solidified lung, communicate more or less freely with a bronchial tube, pectoriloquy is heard. These often pass into each other by insensible gradations; in some cases a cavity, not favorably placed for propagating the sound, giving rise to bronchophony; and in others, a portion of solidified lung favorably disposed for this purpose producing pectoriloquy.

In some cases, however, this increase in the propagation of the sound of the voice is neutralised by the intervention of fluid; for instance, when a portion of one lung is solidified, and at the same time is entirely surrounded by pleuritic effusion. When, however, the chest contains only a moderate amount of fluid, if the lung is healthy, the lower part of it becomes solidified to a certain extent by the pressure of the fluid which prevents its expansion with air. Bronchophony is then formed in it, and becomes perceptible to the ear along the surface of the fluid, where there is not a sufficient quantity interposed to interrupt its propagation to the walls of the chest. But if this portion of the lung be at the same time solidified by disease, less fluid can be contained between it and the costal pleura, and at the same time a stronger vocal resonance is propagated, and consequently bronchophony may exist over the whole of that part of the chest which is in contact with the fluid. The same effect may be produced by the lung being connected to the ribs by loose bands, which not unfrequently occurs.

It has been remarked that the tone of the voice, when heard through the walls of the chest and trachea, is more coarse and buzzing than when it reaches the ear in the ordinary manner through the atmosphere. The same modification is observed in the tone of musical instruments. If the ear, with or without the intervention of a short rod, such as a solid stethoscope, be rested on a pianoforte, or on the sides of a wind instrument, the sounds of the notes are perceived to be more harsh and buzzing than when heard in the ordinary manner. This does not altogether depend on the ear being brought close to the point at which the sound is generated; because if a hollow stethoscope be applied close to the mouth of a wind instrument, but not in contact with it, the intensity of the notes is increased, but their quality remains unaffected.

In accordance with these facts, we observe the quality of tone of pectoriloquy which arises from a cavity in communication with the trachea to be generally clear; and that of bronchophony, which arises from solidification of the lung, to be coarse and buzzing. Between these two extremes there are various gradations, owing to the different states of the lung on which they depend.

When the sonorous waves have also to traverse fluid, as in pleuritic effusion, a broken tremulous character is given to them, in accordance with the experiments of Messrs. Sturm and Colladon. Hence slight *bronchophony* becomes *œgophony*, or the bleating goat's voice, and strong buzzing bronchophony is changed into the coarse nasal *Punch* voice.

Dr. Skoda is of opinion that vocal resonance at the surface of the chest results from consonance of the air in the bronchial tubes, rendered more adapted for such a purpose by solidification of the lung around them. He has thus confounded consonance with propagation of sound.[1] According to this view the air contained in the body of a violin consonates with every note of the gamut, rather than with one particular note. Waiving, however, this point, the exclusion of the sides of the bronchial tubes from any material share in the propagation of the sound of the voice cannot be maintained. All the experiments brought forward by Dr. Skoda only prove that such sounds are best conducted by a tube with solid sides, as has been shown above. He asks why a solid stethoscope is not employed, if the sounds are conveyed in any great degree by the sides of the tubes. The answer to this question will be found in the sixth chapter.

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[1] Prop. XVII.

CHAPTER V.

AUSCULTATION OF THE SOUNDS OF THE HEART.

When the ear is placed on the chest to the left of the middle of the sternum, two sounds are heard, somewhat resembling those which are produced by the utterance of the words *tiff-tac.* After these there is a pause, and then they are repeated. The former sound is synchronous with the pulse of the carotid arteries, and is longer and more muffled than the latter, which is short and clear. The space over which they are heard varies in different individuals, but in health seldom exceeds two square inches.

Now as these sounds are altogether irrespective of the act of respiration, being heard alike during its performance and its suspension, the only movements which can give rise to them are those of the heart; consequently they are called the sounds of the heart, the longer of the two being styled the *first* or *systolic*, and the shorter the *second* or *diastolic* sound.

The following are the movements of the heart which may be supposed capable of producing sound:

1. The rubbing of the heart on the pericardium.
2. The striking of the heart against the ribs.
3. The collision of the particles of blood with each other, and with the interior of the heart and large vessels.
4. The collision between the internal surfaces of the ventricles after the expulsion of the blood.
5. The motion of the different valves.
6. The movement of the muscular fibres of the walls of the ventricles during their contraction and dilatation.

The connexion between these movements and the sounds of the heart has been more or less carefully examined by various persons: in France, by Marc d'Espigne, Ronanet, Bouillaud, Pigeaud, Piorry, Majendie, and others; in Great Britain, by Dr. Hope, Dr. Spital, Mr. Turner, and by Committees appointed by the British Association for the Advancement of Science, in the years 1835 and 1836; that in Dublin consisting of Drs. Macartney, Adams, Kennedy, Green, Hart, Joy, Nolan, Law, and Carlisle; and that in London of Drs. C. B. Williams, Todd, and Clendenning. The experiments of the Dublin Committee were made on calves, and those of Dr. Hope and of the London Committee chiefly on young asses, which had been either stunned, pithed, or poisoned by woorara juice, and in which artificial respiration was kept up. Those of the London Committee[1] and of Dr. Hope, being the most recent, will be chiefly referred to in the following analysis of the movements above enumerated.

1. *The rubbing of the heart against the pericardium* during the contraction and dilatation of the ventricles cannot give rise to any appreciable sounds; because, when the pericardium was removed from the heart in the above-mentioned experiments, the sounds remained unimpaired. Were the two sounds thus produced they would be of the same quality one as the other, although of unequal duration; and therefore sounds of so different a character the one from the other, as the systolic and diastolic sounds of the heart, cannot be thus produced.

2. Neither sound can under ordinary circumstances be produced by the *stroke of the heart against the ribs;* because they were distinctly heard when the ribs and sternum were removed. But it was also found that when the ribs were struck by the apex of the heart, the intensity of the systolic sound was increased; hence it follows that, to use the words of the London Committee, "impulse is not the principal cause of the first sound, but it is an auxiliary and occasional cause, nearly null in quietude and in the supine posture, but increasing very considerably the sound of the systole in opposite circumstances."

3. Neither the *collision of the particles of blood against each other or against the interior of the heart* can be the principal cause of the systolic sound, because it was distinctly heard when the heart was separated from the body, although with less intensity than before its removal. If, therefore, the collision of the blood produces no great amount of sound when forced along by the powerful contraction of the ventricles, it is not likely to

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[1] Reports of British Association, vol. v, p. 261.

give rise to the sound which takes place during the diastole of the ventricles, when the blood is entering them from the auricles, through a very large orifice, with a slower and more feeble current; more particularly as it is well known that this current is often met by blood regurgitating from the ventricles, without any increase of sound taking place. The experiments of Dr. Spital and of the London Committee have proved, practically, that the collision of the particles of fluid against each other very rarely gives rise to sound; a fact rendered highly probable on theoretical grounds, as the obstruction offered by the particles of bodies to each other varies inversely with their mobility, and the consequent facility with which they are displaced.

4. The sounds are not produced by the *collision of the internal surfaces of the ventricles, the one against the other,* as this cannot take place till the blood is expelled at the close of their systole, whereas the first sound was heard to commence with the systole, and the second with the diastole of the ventricles.

5. When one of the aortic or pulmonary valves was hooked up, the second or diastolic sound was replaced by a soft, prolonged hissing, and appeared again on the valve being released. And when the flow of blood was arrested, and with it the action of these valves, the diastolic sound ceased, and reappeared on the current being re-established. From this it follows that *the diastolic sound is produced by the unfolding and tightening of the internal valves, and the stroke of the whole column of the blood against them.* This is further confirmed by the maximum intensity of the second sound being over the seat of these valves, and from its being traceable more easily up the course of the aorta than in any other direction.

But, in like manner as the sigmoid valves are unfolded during the diastole of the ventricles, so are the auriculo-ventricular valves at the commencement of its systole. When, however, the action of these last-named valves was stopped, either by arresting the passage of blood into the heart, or by pushing the sides of the auricles into the auriculo-ventricular openings, the systolic sound was still audible, although fainter than before. Hence it was justly concluded, that the unfolding and tightening of the auriculo-ventricular valves were not the principal cause of the systolic sound of the heart.

6. There remains, therefore, only the *movement of the muscular fibres of the walls of the ventricles during their contraction,* as the cause of the systolic sound of the heart, which was heard after the flow of blood had been arrested.

No sound was heard during diastole in the heart removed from the body, and therefore the muscular sound cannot enter into the formation of the second or diastolic sound.

It has long been known that a sound is engendered during muscular contraction, having a rumbling, intermittent character, but becoming more continuous in proportion to the energy of the contraction. From this Wollaston was disposed to think that muscular action was not perfectly continuous, a conjecture the accuracy of which has been proved by the researches of Mr. Bowman,[1] who has shown that contraction does not take place simultaneously throughout the whole length of a fibre, but in small portions successively, each of which thus becomes wider, and also denser, by the approximation of the discs of the primitive fibrils.

The London Committee conclude that the sound is elicited during the muscular action, by the sudden tension of the fibres,—"the sudden transition of the ventricles from a state of flaccidity in diastole to one of extreme tension in systole." Dr. Williams illustrates this mode of the production of sound by that which arises from the sudden tightening of a piece of linen. But the walls of the heart cannot be compared, in this respect, with a piece of *loose* linen, but rather with one which, being slightly stretched, returns to its first position after having been pushed suddenly into a greater degree of tension; because they return by their resilience to the position they occupied before contraction, the moment that act has ceased. Now if a piece of stretched linen or the sides of an Indian-rubber bottle be quickly and forcibly pushed by the finger, as much or more sound is engendered by its return to its first position as by its departure from it, in obedience to the impulse impressed upon it; unless, as in a drum, a much sharper and stronger blow be inflicted than can arise during the contraction of the heart. As, therefore, no sound is heard during the diastole of the ventricles, when the heart has been removed from the body, it follows that the systolic sound is not engendered by the sudden tension of their muscular fibres. Nor need we be surprised at this, when we consider that the act of contraction destroys, during its continuance, the homogeneity of the fibres, by inducing a state of alternate condensation and rarefaction, so to speak, of their component particles. Now the only other manner in which sound can be produced by muscular contraction is by the friction which takes place between the fibres. This must be very great, owing to the lateral bulges formed by their partial contractions. The moment contraction ceases, these bulges recede from each other, and the whole of the fibres become simultaneously elongated, with little or no friction; and hence the absence of sound during the diastole of the ventricles. The muscular sound is therefore a friction-sound.

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[1] Phil. Trans. of Royal Society, 1840, Part ii, p. 547.

But the London Committee consider the constant systolic sound of the heart to consist of the muscular sound *alone;* a conclusion which cannot be allowed to have been soundly deduced from their observations, as has been shown by Dr. Hope. They describe the first or systolic sound as fainter when the action of the auriculo-ventricular valves was impeded; but, as at the same time the flow of blood through the heart was arrested, they explain this diminution of sound by supposing that the heart acts with less energy when empty than it does when it contracts upon and propels the blood. This is altogether hypothetical; whilst it may be stated, on the other hand, that they have attributed the increased intensity of the sound of contraction of the abdominal muscles over that of the muscles of the limbs to their lying over the cavity of the abdomen. According to this view, the sound of contraction should be louder when the heart is empty than when it is filled with blood. It is indeed difficult to understand on what grounds the action of the mitral and tricuspid valves should be excluded from taking any part in the formation of the systolic sound of the heart, when the whole of the diastolic sound has properly been attributed to a similar action of the aortic valves.

On the supposition that the systolic sound is solely formed by the muscular sound, it is impossible to explain in what manner it becomes modified in certain cases. Thus when the energy of the heart's action is diminished by debility, as in typhus fever, not only does the systolic sound become fainter, but its quality is altered; it gradually becomes shorter and clearer, and thus approaches in character to the diastolic sound. But if the tightening of the auriculo-ventricular valves be allowed to be an element in the formation of the systolic sound, all this becomes intelligible. For as the force of muscular contraction diminishes, and with it the intensity of its friction-sound, a point is reached before the entire extinction of the systolic sound, when that part of it which is produced by the closure of the auriculo-ventricular valves alone remains, and is of a similar character to that of the diastolic sound, although of less intensity, owing to the difference in the situation of the respective valves in relation to the ear, and the different manner in which the blood impinges on them during their closure.

Again it will be presently seen that a bellows-sound may be engendered by the blood being thrown into vibration, by its particles forcibly striking the orifice of the aorta during the systole of the ventricles. Now in certain cases it requires only a very slight increase in the heart's action to produce a bellows-sound; hence it may be concluded that there is a point, a little short of this, when the collision of the blood against the orifice of the aorta strengthens the systolic sound, and in such a case is an auxiliary to it.

It is, therefore, submitted that

The systolic sound of the heart is caused by the friction of the muscular fibres of the ventricles inter se, *and the tightening of the auriculo-ventricular valves; strengthened in certain cases by the impulse of the heart against the ribs, and by the collision of the blood against the orifice of the aorta and pulmonary artery.*

The natural sounds of the heart may be altered in their *intensity* and *quality*, and by the *mixture of other sounds with them.*

Their *intensity* and *quality* are affected by all causes which tend to facilitate or oppose their *formation* and their *propagation.*

One of the most obvious of those which facilitate their *formation* is the energy with which the heart contracts while it retains its normal structure. This is found to be greatly increased above its natural standard by moral emotion, hysteria, inflammation, and the reaction which follows great depletion.

When the heart contracts with *increased* force, all the component parts of its sounds, both constant and auxiliary, are augmented. Thus the muscular friction-sound becomes louder, its intensity varying directly with the force of contraction. The auriculo-ventricular valves are tightened more quickly and forcibly, and so give out a louder flap. The apex of the heart is made to strike the ribs with greater force, and thus the sound of its impulse is increased; and to such a degree in certain cases of hysteria, that the sound thus produced can be heard at a distance of two or three feet from the chest. This occurred in the case of a girl, recently an in-patient of the Birmingham General Hospital. The blood is also more forcibly driven into the aorta, so that a bellows-murmur is thus sometimes engendered. Lastly, the back stroke of the arterial column of blood being proportionate to the impulse it received from the contraction of the ventricles, unfolds the sigmoid valves with augmented force, and the diastolic sound is strengthened.

It is hardly necessary to remark that the sounds of the heart are *propagated* up to the ear with greater intensity than usual in proportion as the walls of the chest are thin and elastic, and as the spongy lung between the heart and the ribs is replaced by solids or liquids, as when the lung is consolidated, or when fluid is effused between the pleural surfaces. In these cases the sounds of the heart are often heard with great distinctness over spots of the chest which they do not in general reach.

If the quantity of blood remain the same, whilst the force of the heart's contraction is thus increased, the duration of it and of its friction-sound is diminished.

When, on the other hand, the force of the heart's action is *diminished,* either by general debility or by disease of its muscular walls, the sounds are weakened in a corresponding degree.

In this case the quality of the systolic sound is also affected, as has been before alluded to; the short clear sound of the auriculo-ventricular valves remaining as long as the blood flows, and after the muscular friction-sound has ceased.

The valves may be so *diseased* as to become thicker and less elastic; and thus the intensity of the sound may be diminished, and its tone may lose much of its clearness. And if the same quantity of blood as usual is propelled, the action, and with it the sound of muscular contraction, will be lengthened.

The sounds too are weakened by being propagated less freely to the surface of the chest, when the distance of the heart from it is increased, as by the effusion of liquid into the pericardium, or the intervention of an emphysematous portion of lung. Here both the impulse is prevented, and, the heart being kept away from the chest, its sounds have to traverse a medium of air or fluid before they reach its surface.

Hitherto the heart has been considered as retaining its normal proportions. But its walls may become thicker or thinner than usual, and its cavities may be enlarged.

When the parietes of the ventricles become *hypertrophied* the increase of their strength is often more apparent than real, owing to a diseased state of their muscular fibres. When, however, they are not thus diseased, the force with which they contract is increased, and with it the amount of muscular friction-sound, and the valvular tension-sound engendered.

But as the walls of the ventricles thicken they remove the inner fibres and also the different valves further from the surface of the chest. Sounds, therefore, arising from their action are less freely propagated to the ear, and thus the increase in the formation of the sounds may be, to a certain extent, balanced by a decrease in propagation.

Besides this the increased thickness and firmness of the bases of the ventricles remove the apex of the heart further from the ribs, and thus in many cases prevents it striking them, and strengthening the systolic sound by its impulse.

The quality of the systolic sound is also affected by this change in structure of the walls of the heart; for whilst the muscular friction-sound may be increased, the valvular tension-sound may be removed further from the ear, and thus the systolic sound may become more muffled and less clear.

When the walls of the heart are *attenuated* the reverse of all this takes place. Less muscular friction-sound is engendered, but it is freely propagated to the ear, and so is the flapping sound of the valves. Thus the systolic sound is not so much weakened as might be imagined, and its tone becomes much clearer and sharper. If the same quantity of blood, however, is propelled, the systolic sound will be prolonged, as in cases of general debility.

When the ventricles are simply *dilated*, a large portion of the heart comes in contact with the ribs and sternum, which are thereby enabled very freely to propagate its sound over a greater part of the surface of the chest. This is illustrated by a person placing on a table first the edge and then the back of a flat watch. In the latter case the ticking of the watch can be heard at a much greater distance along the table by the ear laid on it, than when the edge of the watch alone touched it. The sounds are not rendered louder than usual over the precordial region, because in this spot the heart, when not dilated, already touches the chest; but they are only heard at a greater distance. Occasionally, however, the sounds of the heart are heard clearly at an unusual distance from the precordial region without any dilatation having occurred.

The quality of the sounds will depend upon whether hypertrophy or attenuation of the ventricles be conjoined with dilatation, and will be clear or muffled accordingly. In like manner, when the aorta is dilated, and comes up to the surface of the chest, the diastolic sound is carried up to the ear at this spot with remarkable distinctness in some cases.

Besides the modification of the natural sounds of the heart, certain other sounds are sometimes engendered, varying in quality and intensity from a soft *blowing* murmur to the sounds produced by the action of the *saw* and the *file*. Now in the experiments which were performed on asses, sounds of this nature were made to appear during the systole of the ventricles by pinching in, and thus narrowing, the calibre of the aorta near its origin, and during its diastole, by hooking up one of the sigmoid valves. The effect of the first of these actions was to offer an obstacle to a flowing current of liquid; and of the last to allow a current to flow through an opening of much less size than that of the tube from which it regurgitated. It was hence argued that this class of sounds was produced by the blood being thrown into sonorous vibration by obstacles at or near the orifices of the heart.

We have to inquire then in what manner these sounds are produced and modified by disease.

Now the production of sonorous vibrations in liquid passing from a vessel into a straight tube, as from the heart into the aorta, depends on these circumstances:

1. The direction in which the fluid enters the tube.
2. Its velocity.
3. The nature of the surface of the vessel and tube.

1. *Direction.* When air is blown straight down into a pipe, little or no sound is produced; but when it is blown obliquely across its mouth, a portion of it strikes sharply against its sides and is turned down the pipe, and the column of air within is put into sonorous vibration. The same holds good in respect to liquid entering a tube. In order that it may enter with little sound, the sides of the vessel must converge like a funnel towards the orifice of the tube, as in fig. 4; by which means none of the fluid enters the tube very obliquely.

Fig. 4.

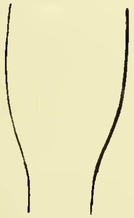


Fig. 5.

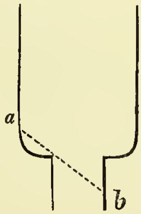
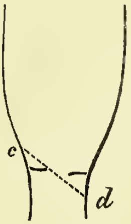


Fig. 6.



But if the base be enlarged as in fig. 5, the fluid from this additional part of the vessel enters the tube at very acute angles with the level of the orifice, as seen by the direction of the line *a b*. It thus enters the tube in a manner favorable to the production of sound.

If the shape of the vessel remain the same whilst the orifice is contracted, as in fig. 6, the same effect results to a certain extent as seen by the direction of the line *c d*.

2. *Velocity.* When the size of the orifice remains unaltered, and the force of the current through it is increased, as for instance by filling up the vessel to a higher level, the increase of the momentum of the flowing fluid will consist altogether in an addition to its velocity, because the size of the column remains the same. And as sound is produced much

more by the velocity of momentum, which gives suddenness, than by its weight, we have a direct increase in the amount of sound, resulting from the increase in the number of particles which strike the orifice of the tube in a given time, and from the additional suddenness of the collision.

If, again, the primary force remain the same, but the orifice be contracted, then if the same quantity of fluid be propelled in a given time, its velocity will be in proportion to the contraction. Practical observations, however, show that this proportion is not preserved, because the same quantity of fluid is not expelled in a given time as it would be if the orifice were not contracted. Still the velocity of the current is increased to a certain extent by the contraction of the tube, and with it the elements of sound. A point, however, is soon reached when this increase of velocity is counterbalanced by the diminished size of the flowing column, and the consequent decrease in the number of particles that strike the tube. For although *velocity* is the principal part of momentum which generates sound, yet its other component part, *mass,* is not altogether to be excluded, otherwise the report of the cannon would be no louder than that of the musket.

Again if the specific gravity of the fluid be diminished, the primary force having less weight of fluid to move, will propel it with increased velocity.

But it must not be concluded that the friction which retards the flowing of liquid from a vessel into a tube gives rise to appreciable sound. A very great resistance is offered to it when flowing from the vessel in fig. 5, by the currents meeting each other almost from opposite directions both in the vessel and in the mouth of the tube, but this collision of the liquid particles against each other has been shown to be very little productive of sound. Dr. Hope has therefore misapplied Venturi's experiments by attributing sound to this cause rather than to collision of the particles of fluid against the orifice of the tube. Again when the interior of long tubes has been highly polished, the flow of liquid is much retarded through them, although but little sound is produced.

3. *The Surface of the Vessel and Tube.* The most direct manner in which a stream of liquid can be thrown into sonorous vibration is by coming into contact with hard and sharp

substances connected with the sides of the tube or its approaches. An abrupt shock is thus communicated to the particles of fluid. If these substances are elastic and project into the stream, they may take on a vibratory action, and thus give rise to a musical note, in the same manner as the vocal cords in the larynx or as the reeds of certain wind instruments.

Now the cavities of the heart are close vessels, the sides of which converge, like those of a funnel, to the orifices by which their contents are discharged. These orifices, particularly those between the auricles and ventricles, are very large in proportion to the size of the cavities from which they lead. Being thus favorably constructed for the transmission of fluids, it is not surprising that, under ordinary circumstances, the blood they contain should be expelled from them with great facility, and without giving rise to appreciable sounds. The force by which it is expelled lies in the muscular contraction of the ventricles, and when this is increased by palpitation whilst the heart retains its normal structure and proportions, the blood is driven with greater velocity into the aorta. When a murmur is thus produced, it must necessarily be short, because the time of the passage of the blood is shortened. Observation, however, shows that a murmur is rarely produced by increase in the energy of the heart's action alone; even when the noise of the apex striking against the ribs is very loud.

When the lining membrane of the heart is inflamed, other causes of murmur are created.

When the ventricles are *dilated*, they become more spherical, and converge less to the aortic openings; as has been pointed out by Dr. Hope. Much of the blood, therefore, enters these openings more obliquely than usual, and thus sonorous vibrations are more easily excited in it, as has been shown. In simple dilatation, which arises from debility and loss of tone of the muscular walls of the ventricles, the force of their contraction is much diminished, while more blood than usual is contained within their cavities. Hence, it must often happen that the formation of a murmur which is favoured by the alteration of the shape of the ventricles, is prevented by the loss of power in their walls, and by the increase in the weight of the blood they have to propel. Consequently, nothing is more common than to find the systolic sound of the heart unaccompanied by any murmur in patients suffering from anemia, as long as they remain quiet, and the heart beats tranquilly; and to hear a well-marked bellows-sound when palpitations are excited. When this sound is thus produced it is prolonged, owing to the diminished velocity and increased quantity of the blood.

If the walls of the ventricles be *hypertrophied,* as well as their cavities dilated, we have the increase in the power combining with the shape of their cavities to produce a murmur. Hence, in such cases it is frequently heard, but not always. In general, however, where no murmur is heard while the heart is both hypertrophied and dilated, its muscular structure is found impaired by disease.

In certain states of the health the specific gravity of the blood is diminished, in consequence of which it is propelled with greater velocity. Not only this, but it thus becomes thinner and less oily, so to speak, and therefore it is more easily thrown into vibration by collision against the solids with which it comes into contact. The blood is doubtless in this state in certain cases of anemia; and if it has to pass out of a dilated ventricle, under the influence of a strong nervous palpitation, a bellows-murmur is a very probable result.

Hitherto the internal surface of the cavities of the heart, as well as their orifices and valves, have been supposed to be in their normal state. It now remains to examine the effect of their diseased states on the production of abnormal sounds.

Substances, from the softness of fibrinous concretion to the hardness of bone, are not unfrequently found both on the valves and on the lining membrane of the heart near them, and the blood flowing over the roughened surface is easily thrown into sonorous vibration.

The valves also become diseased in such a manner that their action is thereby impeded. When thickened or glued together so as to project into the orifices to which they are attached, they narrow them, and thus the velocity of the blood is increased, and it enters the contracted orifice at angles favorable to the production of sound.

Thus a murmur is frequently produced during the systole of the heart by the blood passing through a contracted aortic orifice, and through the auriculo-ventricular openings during its diastole; but less frequently in the latter case, owing to the comparatively slow rate at which the blood flows.

The valves may also be so altered as to allow of the blood regurgitating through narrow openings. Thus in the aorta one or more valves may adhere to the side of the vessel, or they may be shrivelled; in either of which cases they cannot close the aortic opening, and regurgitation and murmur take place in diastole. Or again, they may be glued together into a permanent ring, and thus a murmur is produced both in systole and diastole.

The same may take place in the auriculo-ventricular orifices; but here it may arise from two additional causes, both of very frequent occurrence: the shortening of the cordæ tendineæ, and the dilatation of the orifice, so that its valves cannot close it. But the production of murmur through these orifices is much less frequent than in the aorta. Indeed, when they are not narrowed, and contain no calcareous deposit, the formation of murmur, even during the systole of the heart, when the blood regurgitates through them, is the exception rather than the rule; a remark which the examination of a great number of cases enables me to make. The reason of this is obvious. When the blood regurgitates from the aorta, it re-enters the ventricle with a force equal to that with which it was expelled from it; but when a portion of it regurgitates into the auricle, while the greater part is driven into the aorta, the force of the former is divided with that of the latter, and by so much diminished. Its velocity is further lessened by its meeting a counter-current from the auricle. It is true a collision thus takes place between two opposite currents of blood, but, as has been shown before, the particles of fluid with difficulty excite sonorous vibrations by impinging on each other.

The valves may also be so altered that their free margins may vibrate in the stream. Thus one or more valves may be thickened at their bases so that they cannot fall flat back when the stream passes through them, or, their bases being glued together, all their edges may thus vibrate. In such cases a well-marked musical sound may be engendered. An instance of this may be found in Case L. The same effect may be produced by fibro-cartilaginous vegetations, which sometimes hang like cords into the ventricle from the margins of the mitral valves, and thus float and vibrate in the current as in Case LIII.

The *pitch* of these murmurs varies with the size of the aorta, and the density of the blood, in accordance with the principle enunciated in Prop. XV. It is not affected by the velocity of the current unless, as in blowing into a flute, the harmonic be elicited. When the orifice is contracted the pitch is somewhat lowered, as occurs in the embouchure of a wind instrument.

The *quality* of the murmur depends on the force of the current, and the nature of the opposing parts of the tube. Thus a bellows-sound will become a rasp- or saw-sound by an increase in the velocity of the blood; and a soft opposing surface may only give rise to a bellows-sound, which will produce no thrill sensible to the hand laid on the precordial region, whilst a calcareous projection will produce the saw- or file-sound, and a strong purring thrill. In the latter case the sides of the aorta enter freely into vibration, as is proved by the purring thrill.

The blood may enter the aorta noiselessly, and be there thrown into sonorous vibration by certain abnormal states of the vessel. Thus the aorta may be narrowed at a particular spot, instances of which will be seen in Cases XXIX and XXX. Or it may be dilated, in which case a murmur may be produced by the dilatation, if it take place abruptly, as in fig. 8. In fig. 7 the current diverges but little from its usual course; while, in fig. 8, that part of it which strikes the sides, as at *a b*, is again reflected obliquely across the orifice of the continuation of the vessel. But, as the velocity of the current decreases with the size of the pouch, a limit is soon found, when the blood will strike its sides so slowly and feebly that no sound will result. When it is formed it must be systolic.

Fig. 7.

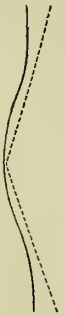
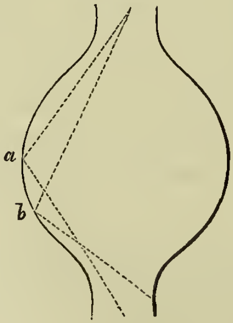


Fig. 8.



An aneurismal sac may communicate with the aorta. If the opening into it be very small, but little blood can enter it, and if it be very large the blood will enter it freely and with little obstacle; in either of these cases little or no sound can be engendered. Again, if the walls of the sac adhere strongly to the frame of the thorax, or if they be thickly lined by layers of coagulum, so that their power of expansion is much limited, then not much blood can enter, and very little sound can be formed. But when the orifice of the sac is of a moderate size, and its walls are elastic, blood may enter the sac during the systole of the ventricle and leave it during its diastole. The sound thus produced will be double.

Sometimes, however, a single sound is heard in the situation of an aneurismal sac. When during systole, it may be that the force of the current may just suffice to produce a sound, during the strong, short systolic action, but the resilience of the sac, during the longer period of diastole, cannot do so. Or the pouch may compress and narrow the aorta. (Case XXIII.) When the sound is only heard during diastole, it is difficult to account for it in any other manner than by supposing a kind of valve to be formed by tough coagulum, which allows the blood freely to enter the sac, but opposes its exit; or else that the margin of the orifice, being very elastic, gives way with the force of the blood entering the sac, and thus projects into it like a funnel; when, however, it projects in a similar manner into the aorta during the reflux of the blood, it narrows it, so that the blood which is flowing onwards is obstructed and thrown into sonorous vibration, and thus a single diastolic sound is produced.

But roughness alone may cause sound, more especially the calcareous scales so often found in disease of the aorta, and this, when joined with dilatation, &c., may give rise to very loud sounds.

All these causes may exist and yet no sound may be produced, owing to the want of sufficient force in the action of the heart by which the current is impelled. Instances of this occur in Cases XXIV, XXV, and others.

CHAPTER VI.

PRACTICE OF AUSCULTATION.

With a view to facilitate the study of auscultation, the chest has been mapped out into different regions by some writers. Occasionally, however, the object in view has not been attained by this plan ; but, on the contrary, the mind of the student has been bewildered by it. In the following pages the locality of each sign is simply denoted by its relation to certain points, such as the vertebræ and different ribs, the sternum, the clavicles, the nipples, &c. &c. No difficulty has been experienced in thus pointing out the exact locality of any particular sound, and the memory is not charged with the task of recalling the various artificial regions.

The sounds which are heard at the surface of the chest, both in health and disease, are classed under a very few heads.

The sounds of RESPIRATION are divided into the *Tracheal, Bronchial,* and *Pulmonary Sounds.*

The varieties of the latter are described by the help of appropriate adjectives, as being intense or feeble, soft or coarse, jerking, stridulous, hollow, blowing, prolonged during expiration, &c. &c.

The dry sounds formed within the bronchial tubes are styled *bass-viol* or sonorous, and *cooing* or sibilous sounds ; and those formed exterior to the lungs *rubbing* and *creaking* sounds.

The liquid sounds are divided into *mucous, muco-crepitant,* and *crepitant* rattles, each being large or small, fine, dry, distant, &c. &c.

Certain other sounds are described exactly as they are heard, viz. *splash, gurgling,* large or small *click, metallic tinkling,* &c. &c.

The forms of resonance of the VOICE at the surface of the chest are called *pectoriloquy* and *bronchophony,* being complete or incomplete, clear, buzzing, nasal, bleating, ringing, &c. &c.

The sounds of the HEART are styled *systolic,* and *diastolic,* being loud or feeble, clear or muffled, extended, distant, ringing, &c. &c.

The valvular murmurs, *bellows-, rasp-sound,* &c. being systolic or diastolic, fine or coarse, shrill, short or prolonged, near or distant, &c. &c.

Percussion. In practising percussion of the chest, it is usual to place the forefinger of the left hand on the chest, and to strike it with the first and second fingers of the right hand curved and held close together. Piorry employs what he calls a plessimeter; a flat substance being substituted for the finger of the left hand, and a kind of hammer for those of the right hand. I have found the ordinary simple method sufficient for all useful purposes. The middle and lower parts of the back of the chest are generally struck with the four fingers of the right hand without the intervention of those of the left hand, the patient having been first made slightly to stretch his back by folding his arms and bending forwards.

Nothing seems easier than thus to elicit sounds from the walls of the chest, which, to a certain extent, shall indicate the state of the organs within them; but, in fact, few things are more difficult: a peculiar elasticity of stroke is required, which long practice can alone give. An adept can, at his pleasure, make a particular spot give out a comparatively clear or dull sound on percussion; this he will effect by allowing the fingers of both hands to remain heavily on the chest after the stroke in the one case, and in the other by quickly removing both hands after a sharp, smart blow has been given, thus deadening or favoring the vibrations of the walls of the chest. The proper method lies between these two extremes.

A novice will be assisted by substituting, for the finger of the left hand, a piece of Indian rubber about half an inch thick, which he must press firmly against the chest. It is hardly necessary to remark, that in comparing the sounds thus elicited from different parts of the chest, the one with the other, care must be taken to place the left forefinger or Indian rubber in a similar manner in both trials; not, for instance, in one on a rib, and in the other over an intercostal space. It is in general advisable to lay the finger across the ribs, at right angles to their direction. Beginners sometimes strike the chest with much greater force than is either agreeable to the patient, or necessary to elicit a proper amount of sound.

If, in any particular case, it is found difficult to decide whether or not the sound is altered from the natural standard, it is advisable, after two or three trials, to leave the patient, and to return, in a short time, with a fresh ear, so to speak.

Auscultation. The sounds caused by respiration, the action of the heart, and in some cases by the act of speaking, are heard at the surface of the chest by the ear placed either directly upon it, or through the intervention of a stethòscope. It is well to habituate ourselves to both these methods, which have each their peculiar advantages. Thus the ear laid directly on the chest detects the exact sound which exists at its surface, unaffected by transmission through any other material. On the other hand, the ear cannot conveniently be placed in the axilla, neither can it ascertain the exact locality of the sounds of the heart and its valvular murmurs with sufficient nicety in all cases; nor is this mode of examination always agreeable to the patient.

The construction of the stethoscope must in some degree depend on the views we entertain of the manner in which it transmits the sounds of the chest to the ear. It has been customary to employ a hollow tube, as it is generally considered that sounds are best conveyed through the air contained within it, which is prevented from diverging by the sides of the tube. Dr. George Budd,[1] whilst employing a hollow stethoscope, maintains that the sounds of the chest are for the most part propagated to the ear by the walls of the instrument; and that the only advantage which results from boring the wood is, that it is thereby made thinner, and thus vibrates more freely. Dr. C. B. Williams, in reply to Dr. Budd, argues that the air within the cylinder must be the principal conducting medium of the sounds caused by respiration, because such sounds, being chiefly formed in air, are by Prop. XIV best conducted by the medium in which they are generated. If we were desirous of listening to these sounds as they issue from the mouth, the hollow, bell-mouthed stethoscope would doubtless prove an excellent ear-trumpet, and would transmit them faithfully up to the ear, through the medium of the air contained within it. But the object of auscultation is to listen *through the walls of the chest.* Before, therefore, sounds formed within the chest can reach either the stethoscope or the ear, they must pass through these walls. The sounds, therefore, which are perceived by the ear, are the vibrations of the walls of the chest; and consequently, by the law above quoted by Dr. Williams, they will best reach the ear by a solid conductor. In respect to the sounds originally engendered in air, a break of medium has necessarily taken place, by their having to pass through the walls of the chest; but we cannot compensate for the loss of sound thus occasioned, by restoring the original medium; on the contrary, we only add to the loss, inasmuch as by Prop. XIII a destruction of sound takes place on *every* break of medium.

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[1] Med. Gaz. 1836.

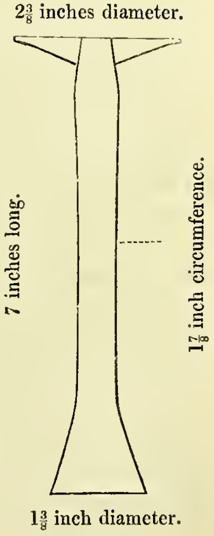
Theory, therefore, indicates the utility of employing a SOLID STETHOSCOPE.

Practical experiments have been adduced on both sides of this question. Dr. Budd states that no sound is heard if the stethoscope be placed as near as possible to the ear and the chest, without actually touching both; and argues that if the vibrations of the walls of the chest were so freely transmitted to the air within the stethoscope as to make it the principal medium of their communication with the ear, the extremely small distance between the surface of the chest and the rim of the instrument before it actually touched it would not allow of sufficient divergence materially to affect the conducting power of the column of air. Dr. Williams brings forward two other facts in support of his view. The one is that the intensity of the sounds transmitted by the cylinder is much diminished by closing either end of it with cork; and the other is, that the sounds are almost lost by boring a hole in the side of the stethoscope, and that they reappear on stopping this hole with the finger. I have frequently repeated these experiments, and have invariably found that although the sounds are weakened, it is not to any great extent. They only prove, therefore, that a certain amount of sound is conveyed by the air within the cylinder, which no person would attempt to deny. In the latter of these two experiments a listener is apt to be misled. The quality of the sounds is altered more than their intensity. When the hole in the stethoscope is stopped, the respiratory sound is accompanied by a deep-toned rumbling, like that of a conch shell; but when the finger is removed, the pitch of the sound is considerably raised, and the rumbling is much diminished. Now this effect is produced equally when the transmitted sound is generated in a *solid* body; as for instance, when the stethoscope is scratched, or placed on a muscle during its contraction; in neither of which cases would any one contend that air was the best conducting medium. In fact, the sounds which are thus altered in quality and intensity, arise from the vibrations of the column of air, excited by those of the sides of the cylinder, and consequently they vary in pitch with the length of such column, as the notes of a flute. In a similar manner, the vibrations of a violin string are communicated to the sounding-board, and from thence to the air within the instrument. Consequently, if the sides of the stethoscope be made very thin, the intensity of their sonorous vibrations will be increased, and the sound will be further augmented by the vibrations of the aerial column within. But the object of auscultation is to have the same sounds as are heard by the naked ear placed on the chest faithfully conveyed by the stethoscope, unaltered either in intensity or quality by transmission through this instrument. It is also stated by Dr. Williams, that if the stethoscope be tilted in the slightest possible degree, the sound is no longer transmitted to the ear, in consequence, as he supposes, of the air being thus allowed to diverge. But it must be remembered, that when the stethoscope is but very slightly tilted, nine tenths of its rim are removed from contact with the chest, and thus the greatest portion of the solid material is prevented from receiving vibrations from its walls.

Some years ago, before I saw Dr. Budd's communication, and, I believe, before it was published, it occurred to me to make trial of a solid stethoscope, with a view of ascertaining whether it would in practice fulfil these theoretical indications. I was pleased to find that it conveyed more of the pure sound which the ear distinguishes when laid on the chest than a hollow stethoscope. The latter would convey sounds of greater intensity, but of a quality which varied with the substance and material of the instrument; being in every case different from the sound perceived by the ear alone. The reason of this seems obvious. The vibrations of the wallsof the chest, however excited from within, are best conducted by a solid material of a similar molecular construction to their own. Thus a solid, bell-mouthed stethoscope, receives the vibrations of the chest over the whole of its base, and not merely on its rim, as in the case of the hollow instrument. Being thus received, they are reflected to and fro from the surrounding air up to the ear, just as aerial vibrations are reflected from the solid sides of a tube.

I have not only thus made use of a solid stethoscope for nearly twelve years, but, having placed several of them in the hands of different gentlemen, I have had the satisfaction to find that, in most cases, they have been preferred to the hollow stethoscope. I would particularly mention the name of my friend, Dr. Watson, late Professor of the Practice of Medicine in King's College, one of the most accomplished and skilful physicians of the age, who has made use of this kind of instrument, in preference to any other, for some years past. It is made of light cedar, the grain of which runs from end to end. The earpiece is slightly hollowed out, and the stem is tapered off to receive it, in order that it may always fit tightly. The following diagram represents a solid stethoscope on a reduced scale.

Fig. 9.



When the *respiratory sound* is faint, the longer the ear rests on the chest the better it will appreciate it; but when there is a doubt about the existence of *vocal resonance,* the ear should be withdrawn for a few minutes and then replaced, otherwise confusion arises.

When the student commences the practice of auscultation at the bedside, his attention should for some time be confined to the sounds of the healthy chest, before it is directed to the modifications induced in them by disease; just as the study of anatomy should precede that of pathology.

When he has once become thoroughly conversant with the natural sounds, patience and perseverance are alone required, in order that he may gain the power of detecting those which result from disease.

And yet how seldom is excellence, I had almost said mediocrity, in the practice of auscultation attained! There are many reasons for this.

Some are deterred from its study by the belief that a musical ear is indispensable for success. This is an erroneous impression. One of the best auscultators of his age I ever knew was a pupil of the Birmingham General Hospital, who has since risen to great eminence, and who had no ear for music.

Others confine their studies to the closet. With no higher aim than to pass the requisite examination for their diploma, they learn a long vocabulary of sounds, and commit to memory certain combinations of them, which are said to exist in particular diseases; and here they stop. They never learn to practise auscultation.

Some, again, having advanced to a certain point, become lost and bewildered amidst the multitude of sounds which the subtle refinements of some authors have caused them to describe; and thus they give up the pursuit in despair.

There is a certain class of students, however, who devote themselves with ardour to the practical study of auscultation, and become expert in detecting the physical signs of thoracic diseases. Encouraged by the enthusiasm of some writers on the subject, they imagine that auscultation can teach them everything connected with such diseases, and will lead them to the adoption of a proper line of treatment, irrespective of the careful study of each case in its individuality. One of this class, on being called to visit a sick person, and having ascertained the existence of pneumonia, would probably at once place him under a course of tartarized antimony, and, knowing the extraordinary efficacy of this drug in such cases, would confidently look for a speedy recovery. Watching closely the progress of the pneumonia, he would be much surprised at finding his patient rapidly sinking, at the moment when, judging from the state of the lungs, he might have expected to see him approaching convalescence. Had he not overlooked the fact that pneumonia was, in this instance, a secondary affection, which had arisen in the course of typhus fever, he would have directed his chief attention to the general state of the patient, and having sustained him with tonic and stimulating remedies, a fatal termination would probably have been prevented. Sooner or later such persons become undeceived. They then either run into the opposite extreme, and renounce auscultation altogether; or else, profiting by the past, they continue to employ it in conjunction with other modes of investigating disease, without allowing it an undue ascendancy; and thus, at length, they become sound and safe practitioners.

An attempt has been made in these pages to remove some of the difficulties which thus beset the path of the student. It has been shown that a musical ear is not required, and at the same time the different sounds of the chest have been simplified and reduced to as small a number as possible. The danger which arises from over estimating the capabilities of auscultation has been pointed out; and the unsoundness of certain general rules for diagnosis which have been founded on it has been exposed. Every opportunity, also, has been seized to inculcate the necessity of studying each case in its individuality, and in all its bearings; and a certain number of cases have been recorded which may serve for the purpose of comparison with those under treatment.

But were all these difficulties removed from the path of practical auscultation, this would not meet the case of those who never attempt to enter on it. What splendid opportunities for practical study occur in the wards of many of our hospitals! and yet how often are they left unimproved! and how frequently does the physician painfully feel that he is unable fully to perform one of the most important and pleasing duties of his office, the instruction of the student in the art of applying the various branches of knowledge to the discovery and treatment of disease! This in some degree arises out of the present system of medical education. Compelled to attend lecture after lecture during a great part of the day, the student has not time to devote both to medical and surgical practice at the Hospital, without exercising such an amount of self-denial as few are disposed to submit to. This evil might, to a certain extent, be remedied by causing the lectures on medicine and surgery to be delivered at the hospital, and by making them consist, in part, of examinations of each pupil in rotation, and in presence of the whole class, on certain cases in the wards which he had previously been directed to watch; and also by conducting a part of the examination for diploma at the bedside, when the examiners would be enabled, among other things, to test the practical knowledge of auscultation possessed by the candidate.

Were every facility and encouragement, however, thus afforded for the practical study of auscultation, excellence would still, as in almost every other department of science, be confined to a few; but all might acquire such an amount of knowledge as would enable them to recognise the existence and nature of thoracic diseases under ordinary circumstances.

CHAPTER VII.

FORMATION, TERMINATION, AND CAUSES, OF THORACIC ANEURISM.

FORMATION.

The general anatomical characters of thoracic aneurism are so well understood at the present day, that it would be useless to enter on an investigation of them. There are, however, some points connected with their mode of formation, and the causes on which they depend, which have not as yet been clearly made out.

It is generally admitted that there are three principal forms of aneurism of the thoracic aorta.

Dilated aneurism, in which all the coats of the artery unite in forming the walls of the pouch throughout its whole extent.

Sacculated aneurism, formed by the cellular coat and neighbouring organs, which the blood has reached through a perforation of the inner and middle coats.

Mixed aneurism, in which the walls of the pouch are partly formed by all the coats of the vessel, and partly by the cellular coat and contiguous parts. Thus the second form has been engrafted on the first by the perforation of the inner and middle coats forming the walls of the dilated aneurism.

The existence of this latter form was denied by Scarpa, but although it has been clearly demonstrated, yet it is probable that many cases of the pure sacculated form have been mistaken for it. Thus in some of the cases about to be described, the pouch appeared at first sight to be of the mixed form; a closer examination, however, showed that the extent of the middle coat, which entered the sac and terminated with an abrupt edge, very nearly corresponded with the size of the aperture into the aorta. Now had the pouch been formed in the first instance by dilatation of all the coats, the extent of the middle coat in the sac must have exceeded the area of the orifice. Supposing, however, the formation of the sac to have been preceded by perforation of the inner and middle coats, if the latter adhered closely to the cellular coat, it would be dragged into the sac formed by the dilatation of the outer membrane. But if these two coverings were not closely united, the cellular coat would be for some distance dissected off the middle coat; which for a time would stand as a kind of diaphragm between the aorta and the pouch. Sooner or later, however, it would yield to the outward pressure of the blood, and be forced against the inside of the mouth of the sac, and probably become agglutinated to its walls by lymph. In either case the result would be a sacculated aneurism, having its orifice lined for a certain distance proportioned to its size by the middle coat of the aorta. It may be argued that the inner and middle coats might have originally extended further into the sac, but had been subsequently destroyed. In that case the raised edge of the middle coat would not have been so distinctly marked, nor would its extent have been so exactly proportioned to the size of the orifice in so many cases.

In some of these cases a thin delicate membrane, apparently continuous with the inner coat of the aorta, may be seen entering the aneurismal pouch, and lining it for a considerable distance. The great extent of surface it thus covers in certain cases proves that in them it cannot be the inner coat of the aorta, which was long since shown by Hunter and Sir E. Home to be never very extensible. Bizot[1] has shown that this membrane is not a continuation of that which lines the aorta, although its point of union with it cannot be detected with the naked eye. It was seen in Cases VIII, XIII, XIV, and others. In Case XIII, however, it not only lined a portion of the sac, but completely invested a large laminated coagulum, and was therefore formed subsequent to the deposition of fibrin in the sac. In Case XI a membrane of this kind closed the orifice of a small aneurismal sac of the size of a hazel nut, which sprang from the arch of the aorta, and was completely filled with layers of dense fibrin. Viewed from the inside of the aorta, the orifice looked like a small gray-coloured depression, covered with the inner coat. When these membranes are submitted to microscopical examination, their nature is at once revealed.

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[1] Mémoires de la Société Médicale d'Observation. Paris, 1837, vol. i, p. 339.

They are found to be totally devoid of epithelial cells, which abound on the inner membrane of arteries, and to possess all the characteristic appearances of fibrinous films, a species of delicate false membrane, which undoubtedly they are.

It cannot therefore be deduced from the existence of such a membrane lining an aneurismal sac, that its walls were originally formed by dilatation of all the coats of the vessels from which it sprang.

For the same reasons we cannot allow the correctness of the inference drawn by Dupuytren, Brechet, and others, from certain cases that the inner membrane is sometimes forced through a perforation in the middle coat, and thus made to form an aneurismal sac by a kind of hernia. It is probable that the false membrane above described has been mistaken for the inner coat of the artery, as pointed out by Bizot.

Mr. Hodgson has inferred the previous existence of the inner coats in aneurismal sacs from the fact of atheromatous and calcareous deposits having been found in them.[1] "This circumstance," he remarks, "appears to me to prove that the coats of the artery compose the disease, for it is the internal coat in which calcareous matter is deposited, and, if we find that substance in all parts of the sac, it appears fair to infer that the internal coat enters into its composition, inasmuch as that identity of disease indicates identity of structure." This observation of Bichat, quoted by Mr. Hodgson, does not apply to the case in point. For calcareous deposit is not confined to the arterial tunics, but is often found in false membranes and imperfectly organized formations on the pleura and pericardium, and even in fibrinous concretions in the cavities of the heart and in tubercular matter. It may therefore be deposited in aneurismal sacs without the internal or middle coat having ever formed a part of them. Nor is it more unlikely that atheroma should be deposited in a similar manner than that tubercular matter should be found in imperfectly organized false membranes.[2] Bizot has detailed a case in which a moderately sized sac was completely lined with atheroma, over which was spread a delicate false membrane. The middle coat terminated with short raised edges around the orifice of the sac. I have seen similar cases.

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[1] Diseases of Arteries and Veins, p. 46.

[2] Op. cit. p. 342.

It may be inferred, therefore, that mixed aneurisms are not of such common occurrence as some writers have supposed, and that the existence of an aneurismal sac formed by the inner coat of the artery, passing through a perforation of the middle coat, is very doubtful.

It is a matter of question whether an artery in a healthy state can ever be dilated. No amount of force in the heart's action would seem capable of producing such an effect, or it would surely have been seen in Case XXXVII; but, although the force of this action was immense, no trace of dilatation was perceptible; on the contrary, the aorta was rather below the normal size. Nor will constriction of the aorta produce this effect, at least to any extent, otherwise it would have occurred in Cases XXIX and XXX. Mr. Hodgson, in reference to an uniform dilatation of the aorta, asks, "may it not in some cases be owing to a state of the fibrous or middle coat not unlike paralysis, for I have seen the vessel morbidly dilated without disease of its structure?"[1] I have also seen the dilated vessel apparently without disease, but never failed to detect the presence of atheroma in its middle coat by aid of the microscope, since my attention was drawn to this point by Mr. Gulliver's paper.[2] But were the vessel healthy, paralysis would only affect its insensible vital contractility, by which means it might be prevented from contracting on a diminished column of blood, but its dilatation could only be occasioned by a loss of the elasticity by which it returns to its usual size after distension.

In respect to the rupture of the inner and middle coats of a healthy vessel, though this was artificially produced by Mr. Hodgson with a ligature, yet he considers that no exertion from within could produce such an effect, nor any violence from without, that did not at the same time rupture the neighbouring parts. The two following cases prove that the heart may be ruptured whilst the pericardium remains unbroken; and that the inner and middle coats of the aorta, and of the auricle of the heart may be lacerated without the outer coat being injured.

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[1] Op. cit. p. 51.

[2] Med.-Chirurg. Trans. vol. xxvi.

Case I.

Laceration of the endocardium and muscular fibres of the left auricle of the heart.

A female, æt. 90, was run over by a cart within a few yards of the Birmingham General Hospital, to which she was immediately carried, and died in half an hour.

Inspection. The sternum was transversely fractured an inch and a half above the ensiform cartilage, without the periosteum being lacerated. The fourth, fifth, sixth, and seventh true ribs on the right side were fractured about an inch from their junction with their respective cartilages. The exterior surface of the pericardium had a healthy appearance, but on opening its sac, a patch of ecchymosis was observed on the posterior surface of the right auricle. At the interior of the auricle which corresponded to this patch, a laceration was observed an inch and a half in length, having irregular edges and extending quite through the endocardium and muscular coat. Through this wound a small quantity of blood had passed, and thus gave rise to the ecchymosis observed. A small ecchymosis was also perceived under the reflected pericardium, opposite to that on the auricle.

Case II.

Laceration of the inner and middle coats of the aorta.

A groom, æt. 54, was running out a horse for sale, when he was suddenly struck on the chest by the shaft of a cart. He was immediately taken to the Birmingham General Hospital, a distance of one mile, and on his arrival there he was found to be dead.

Inspection. The sternum was transversely fractured between the second and third ribs. The second, third, and fourth left costal cartilages were cracked through. The pericardium contained two pounds of blood, which had escaped from the heart through an aperture in the appendix of the left auricle half an inch in length. A laceration an inch and a half long was found in the aorta between the orifices of the left common carotid and left subclavian arteries. It extended through the inner and middle coats of the vessel. A small quantity of blood was extravasated for some distance around it under the cellular coat which was uninjured.

I was present at the examination of the bodies in both these cases, but the notes have been abridged from those taken by Mr. Alfred Baker, at that time house-surgeon to the hospital, who kindly placed them at my disposal.

Supposing such laceration not to have been immediately followed by death, it is not probable that, even in that case, aneurism would have resulted; for the experiments of Dr. Jones and Mr. Hodgson render it likely that lymph would have been thrown out, and that the edges of the wound would have been reunited. This point, however, is of no great practical value, because it will be seen that more or less of structural change is always found in the coats of the aorta when it is the subject of aneurism.

TERMINATION.

Thoracic aneurism generally proceeds to a fatal termination; which either results from the escape of blood through a perforation of its walls, or from pressure on some vital part near it. Its progress, however, is influenced by several circumstances connected with its *situation, the state of its walls,* and *the nature of its contents.*

Situation. Aneurisms which arise either from that portion of the aorta which is within the sac of the pericardium, or from that which lies between the left bronchus and the diaphragm, usually run their course without materially affecting the neighbouring organs. Those which spring from the root of the aorta seldom if ever attain a size larger than that of an egg, before they burst into the pericardium, in consequence of that membrane, which constitutes the outer coat of the vessel in this spot, being very thin and inextensible. A case is recorded by Dr. Smith,[1] in which an aneurism in this situation had attained a considerable size; but this is of very rare occurrence. Cases of this kind are recorded by Morgagni, Cooper, and others. I have myself seen four such; one of these was Case III, and the other three were very similar. Aneurisms, therefore, which arise within the sac of the pericardium, do not in general attain a sufficient size to cause them to press upon the neighbouring parts.

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[1] Dublin Journal, vol. ix, p. 423.

When they occur at the other extremity of the thoracic aorta, below the left bronchus, they occasionally attain a very large size, but in that situation they only encounter the bases of the lungs, the apex of the heart, and the œsophagus, parts which allow of considerable displacement, without any fatal consequences.

The intermediate portion of the aorta, however, is surrounded by many important parts, so that aneurisms arising from it can hardly fail to interfere with the functions of some of them. Something, however, here depends on the exact part of the vessel from which they spring, those from the anterior side gaining the surface of the chest, and reaching a tolerable size without materially affecting neighbouring parts; whilst a very slight dilatation of the posterior portion interferes with the trachea, œsophagus, &c.

Walls. The constant tendency to attenuation and rupture of the walls is not unfrequently, to a certain degree, counteracted by an inflammatory process, in which the walls are thickened by the deposition of lymph.

Unfortunately, whilst the walls are thus strengthened, they are at the same time made more solid and less yielding, so that they are less able to accommodate themselves to a neighbouring part, when they reach one by dilatation. The effect of this was very strikingly seen in Cases XI, XXII, and XXV. In XI and XXV the sac adhered to the vena cava, and so compressed it that extensive œdema of the head, neck, and upper extremities supervened. In XXII the sac yielded, and the vena cava was found lodged in a deep groove, and was thereby protected from pressure, and there was no trace of œdema.

So again, on the aneurismal pouch touching a neighbouring part, adhesive inflammation is often set up between them, in the same manner as the stomach and intestines are sometimes glued together. Whilst perforation is thus often prevented, the sac is sometimes so fixed to some vital part that it cannot leave it and increase in some other direction, where it would produce less inconvenience. This was observed in Cases X and XXV, where the sac was fastened down to the trachea; in Cases XI and XXV, where it adhered closely to the vena cava, and in Case VIII, where it was bound down to the spine, and erosion of the vertebræ followed. Hence the distress produced by aneurisms is by no means in proportion to their size, but, as has been shown by Dr. Law, rather in an inverse ratio; because those which are not tied down become large, and reach the surface without materially deranging the functions of other parts.

Contents. Mr. Hodgson was the first clearly to demonstrate the manner in which the growth of aneurism is, in certain cases, checked by the coagulation of the blood within the pouch. His researches on this point are characterized by an accuracy of observation and soundness of deduction rarely met with.

This process depends, in some degree, on the amount of inflammation of the walls, as it is well known that inflamed surfaces tend to cause the coagulation of blood in contact with them; and not only this but the layers of fibrin which are thus deposited in the interior of an inflamed sac are agglutinated to it by lymph. For want of this, perforation may take place after a sac has been filled with coagula; although Mr. Hodgson considers that a "sac filled with coagulum cannot prove fatal by rupture."[1] This was seen in Case XXIV. Had the clot been firmly glued to the walls of the sac, the fatal result would not have thus occurred.

When the sac encroaches on the lungs, before the outer coat is destroyed, the lung is often prepared to become a boundary wall to the cavity, by the coagulation of blood in its parenchyma, and by a deposition of lymph. This was seen in Cases VIII, XVIII, and XIX.

Coagulation also depends, in some degree, on the force of the circulation, and is consequently favored by feebleness of the heart's action, &c. The circulation of blood in the sac itself is much affected by the amount of elasticity in its walls. Thus the thicker and more inelastic are the walls, the less will they yield to the force of the systole of the heart, and the less blood will enter and leave the sac. The same result will take place, when the sac having been gradually enlarged, at length meets with and becomes united to a firm resisting substance, such as the sternum or ribs, whereby its power of expansion is limited.

Coagulation also, or, at least, the nature of the clot depends on the quality of the blood. If the blood be impoverished and deficient in solid constituents, the clot will be soft and gelatinous, and little likely to adhere to and strengthen the walls of the sac.

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[1] Op. cit. p. 118.

Mr. Hodgson has very well described the method by which an aneurismal cavity is thus obliterated. Being filled with coagula, the tendency to dilate from the outward pressure of the blood is arrested. The clot then becomes smaller and drier by absorption of its fluid parts, and the walls of the sac contract upon it, till at length only a solid fibrous tumour remains. Two instances are given (Cases xx, xxi) in page 119 of his work. In both these cases, however, death was produced by pressure on the trachea; so that solidification of the tumour, although it prevented perforation, caused increased pressure on the trachea and death.

As long as the inner coat of the artery remains entire, no clots are deposited on the interior of the aneurism. The various stages of fibrinous deposits may be traced in several of the cases which follow.

In the great majority of cases death ensues, and that in the manner above stated, through injury to the neighbouring parts, or from escape of the blood either externally or into some internal cavity.

Sometimes the action of the heart ceases from its inability to force the blood through a large dilated aneurism, the contents of which oppose the free passage of the blood into the descending aorta, and the vessels which open from the arch. I have not witnessed such a mode of death myself, unless it was thus caused in Case xiii.

In the following cases, and in the writings of different authors, a great variety of instances may be seen of death produced by pressure on different organs, and by the escape of the blood into various cavities, or through the integuments.

It is difficult to imagine that the efforts of nature to ward off death should for a time succeed after the aneurismal sac has actually burst into a cavity of the body; and yet Cases vi, viii, ix, xxiii, and xxiv prove that it is by no means uncommon for the orifice to become plugged up by portions of the coagulum in the sac. In Case xxiii life was even preserved for many months after this had occurred, and in Case ix for some days, also in Cases vi and viii for a considerable time.

CAUSES.

Dilatation or perforation of the aorta must arise either from a very great increase in the force with which the blood is impelled into it by the heart, or from a diminution of elasticity and loss of substance of its inner coats. The middle coat, however, is so strong, tough, and elastic, that it is impossible to conceive that its permanent dilatation or rupture can be produced by any effort of the heart, so long as the vessel continues in a healthy state. We have seen accordingly that neither excessive hypertrophy of the left ventricle, nor contraction of the aorta at particular points, will produce such effects.

Diminution of elasticity and loss of substance alone remain, therefore, as the proximate causes of aneurism. These can only result from a morbid alteration of the arterial coats, or a total or partial displacement of their proper substance by matter possessing less of elasticity and cohesion. Alterations of this kind are therefore constantly found resulting from a deposition of horny patches, atheroma, and calcareous concretions.

Horny patches. The inside of the arteries sometimes presents a rugged and uneven appearance, caused by a substance somewhat resembling softened horn, and disposed either in patches or around the whole circumference of the vessel, in some places being opaque, and in others more or less transparent. Andral considers that this appearance is produced by a deposit between the inner and the middle coats; and Mr. Hodgson represents it as resulting from a thickened state of the inner membrane, which he compares to the peritoneum of an old herniary sac. Bizot, however, has shown that it arises from a deposit on the free surface of the inner coat. He has traced the substance deposited passing by insensible degrees from the consistence and transparency of jelly to the opacity and almost the hardness of cartilage, and this in an exact ratio with the age of the subject examined. In three cases in which the patients were suddenly seized and quickly carried off by an acute attack, he found the matter in its softer state and of a rosy colour lining a great part of the principal arteries. He has shown that these patches in an early stage of their development, before they have acquired hardness and opacity, may be removed with the inner membrane, which may be thus traced under them. They are often covered by a delicate film, similar to that which has been seen lining aneurismal sacs. Bizot considers this deposit to be a secretion from the inner coat of the artery acted on by inflammation; being, in fact, coagulated fibrin, resulting from what Vogel denominates fibrinous dropsy. Patches of a similar kind are sometimes found on the pericardium covering the heart, and on the lining membrane of the left auricle, in which situation they are generally allowed to be the result of inflammation. Bizot states that they never undergo osseous transformation, as asserted by Morgagni, Haller, Beclard, and Andral. In confirmation of this, it may be remarked that calcareous deposits in the aorta are not true bone, but merely concretions, which have not been formed from cartilage by cellular development; nor are these horny patches true cartilage. Viewed under the microscope, they are seen to consist of an amorphous mass, more or less transparent, and rendered still more so by acetic acid, which contains some minute granules and some pale cells without nuclei. In some cases no cells can be discovered; in others there is a trace of fusiform cells, showing the tendency towards the formation of areolar tissue.

Repeated examinations have convinced me of the accuracy of Bizot's researches, and it may therefore, I think, be concluded that these horny patches are imperfectly organized false membranes, deposited on the free surface of the inner coats of arteries.

The horny patches are not found in all cases of aneurism; when they reach an advanced stage, they cause the destruction of the inner coat of the vessel; but as they do not invade the middle coat, they cannot be considered materially to diminish the elasticity of the artery, or to favour its perforation, unless in conjunction with some other cause.

Atheroma is of a brownish yellow colour, and of a consistence varying from that of pus to cheese, or boiled white of eggs. When joined with areolar tissue, it is in certain cases still firmer, and has been then called steatoma; a term, however, which has been occasionally applied to the horny patches by some writers. It originates between the inner and middle coats, and in the substance of the latter. Its changes very much resemble those of tubercle, and consist of softening and of ossification, as it is commonly called. When it softens, it produces ulceration of the middle membrane, followed by rupture or destruction of the inner coat. It not unfrequently thus burrows under the horny plates.

This substance is constantly found in aneurisms, and as its tendency is to destroy the elasticity of the artery it invades, and to favour its rupture, it is universally allowed to be the most constant proximate cause of aneurism.

Gluge found atheroma to be chiefly composed of fat, and Cruveilhier discovered cholesterine crystals in it. This was confirmed by Hasse, and also by Mr. Gulliver, who was not aware, however, of the researches of his predecessors. Vogel has described a mass of atheroma as consisting, "1, of many tubular colourless crystals of cholesterine of the ordinary characteristic form, rhomboidal tablets; 2, of many irregular amorpho-granular masses, which did not dissolve in water, but were soluble in alcohol; after the evaporation of which they again thickened into amorphous brownish clots, probably fat. Besides these elements and some few fat-globules, nothing was present."[1]

Calcareous concretions. Instead of softening, calcareous grains are sometimes deposited in the patches of atheroma, which, coalescing, form crusts and bony-looking scales. They are described by Valentin[2] "as organized calcareous deposits, the earthy bodies being deposited as round, granular, or irregular compact bodies (with prolongations radiating in all directions), within a pellucid, more or less lamellated, and finely granular organic texture." It is hardly necessary to insist on the difference between these concretions and natural bone. They are principally composed of phosphate, with some carbonate of lime.

Now atheromatous deposit cannot be a local affection, for it is not only found throughout the whole of the arterial system, but occasionally in the lungs, where it would seem to have replaced tubercular matter; and I have also found it in the glands of the gastro-hepatic omentum associated with calcareous concretions. Dr. George Johnson[1] has shown that the arterial tunics in a healthy state contain some minute quantities of fat, so that the affection under consideration consists in an excessive deposit of fat in tissues which, in a state of health, are sparingly supplied with it; a process closely resembling that which has been demonstrated by Mr. Bowman[2] in fatty liver, and by Dr. George Johnson in the uriniferous tubes of the kidney in Bright's disease; and with both which diseases Dr. Johnson has found atheroma very frequently associated. He therefore concludes that "these diseases are related to each other as joint effects of one common constitutional cause."

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[1] Vogel's Pathology, by Day, p. 584.

[2] Hasse's Pathological Anatomy, by Dr. Swaine, Sydenham Soc. Edition, p. 79.

It has been shown by Becquerel and Rodier[3] that the amount of free cholesterine in the blood varies very much, and that it is greatly increased at that period of life at which we shall presently see there is also an increase of atheromatous deposit. Upon this Vogel remarks, that "the augmentation of this substance in the blood is probably connected with an increased separation of it in the various parts of the body, and that a similar increase may take place in young persons in consequence of a morbid process."[4]

Both Andral and Lobstein think this state of the constitution is closely allied to that which exists in arthritis. There is, however, a great difference between the chemical composition of urate of soda and atheroma, and so also is there between the latter substance and tubercle; and yet there are some striking points of resemblance between the modes in which each of these substances is deposited in the tissues.

In the present state of our knowledge, we can, I think, go no farther than to infer that the deposition of atheroma is a process of abnormal nutrition, depending on a peculiar state of the constitution, which may be denominated the atheromatous diathesis. It is very probable, however, that future investigators will discover that the same state of the constitution gives rise to other forms of disease.

The diathesis being once established, the ulterior changes of the atheromatous concretions may be modified by a variety of circumstances tending to hasten or to retard the rupture and dilatation of the artery.

[1] Med.-Chirurg. Trans. vol. xxix.

[2] Lancet, Jan. 1842.

[3] Comptes rendus, 1844, p. 1085.

[4] Op. cit. p. 340.

Much must of course depend on the amount of constitutional derangement; but something also on the quantity of the general nutrient fluid, and the consequent moisture or dryness of the exudation. This must bear some proportion to the state of the system generally, and to the activity of the circulation. Moisture of the exudation might tend to produce softening and ulceration, whilst dryness would probably give rise to the cheesy state. It is probable that a mixed plasma is thrown out, as supposed by Vogel,[1] who remarks, "In this exudation, two formative processes are simultaneously going on: an organization of the fibrin, and the formation of concretions, consisting generally of earthy salts. The product of this formation consists chemically of two distinct steps, one relating to the conversion of the fibrin, and its modifications into areolar tissue, granular cells, typhic deposits, and tubercular matter; the other to the constituents of concretions, as the salts of lime and magnesia, the urates, fat, &c. The individual constituents of either group may assume a vicarious position; moreover, the whole of the first group may assume the place of the second, and conversely, the second may replace the first, so that the one is subordinate in the same degree as the other predominates." When, therefore, rapid ulceration takes place, the former process predominates; and when the matter remains dry, or is succeeded by calcareous deposit, the latter takes the lead.

Other causes may also concur with atheromatous degeneration to favour dilatation and rupture. Some of the effects of inflammation on the lining membrane have been seen; the elasticity and cohesion of the fibres of the middle coat may possibly be also diminished from the same cause. But inflammation of arteries is generally attended by the effusion of plastic lymph, as is proved by the experiments of Dr. Jones and Mr. Hodgson, before alluded to. Whilst, therefore, it might on the one hand diminish the elasticity of the middle coat to a certain extent, the tendency to dilatation or rupture would probably be counteracted by the effusion of lymph between the middle and outer coats.

Again, the force with which the blood is propelled into the aorta, may be increased by nervous palpitations under the temporary influence of the passions, and from sympathy with diseased organs; or else by hypertrophy of the left ventricle of the heart. A violent shock also may cause the rupture of a diseased vessel, and probably did so in Cases XIV and XX.

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[1] Op. cit. p. 387.

Such are some of the causes which may be supposed to favour the formation of aneurism when atheroma has been deposited in the coats of an artery. The influence of two further causes has been ascertained by actual observation. These are *age* and *sex.*

Age. An examination of cases recorded by different writers proves that aneurism prevails especially between the ages of forty and fifty. But it will presently be seen that the atheromatous diathesis increases with age, seldom being marked under forty. It would appear, therefore, that above fifty, aneurism decreases, whilst atheroma increases. The following observations may perhaps help to explain how this occurs. Assuming atheroma to be the constant proximate cause of aneurism, its rarity under forty accounts for the rarity of aneurism under that age. When, however, it is once deposited, the younger the subject the greater would be the tendency to the formation of aneurism. For this depends not so much on the amount of atheroma as on its tendency to soften; now the younger the subject, the more active would be the circulation, the moister the exudation, and the greater the tendency to ulceration. In old age the reverse of this takes place, the circulation is retarded, the quantity of the general nutrient fluid is probably diminished, and the atheromatous patches are dry, or are soon converted into calcareous concretions. By the same law, the softening of tubercular matter takes place with less and less rapidity as age advances, so that the older the patient the more chronic is the march of the disease. Again, the nerves are more excitable, and the muscular power is greater in middle than in old age; hence nervous palpitations are more easily excited, and the action of the heart is more energetic. At this period of life, more violent exertions are usually engaged in, and falls and shocks of different kinds are of more common occurrence than in later life. On the other hand, however, Bizot has shown, that the size and thickness of the heart gradually increase up to extreme old age, but probably not in a greater degree than is necessary to compensate for the diminished elasticity in the arterial system.

Sex. The influence of sex is most marked, aneurism only occurring in one female to nine males, or thereabouts. Bizot, however, found very little difference in the amount of atheromatous deposit in the two sexes. Now, the only difference between the sexes which could be supposed to have any influence on the formation of aneurisms, is that which exists in respect to habits and modes of employment. In this view of the subject, it would be very interesting to know the ratio in which aneurism is found in the two sexes in those countries where females undertake laborious employment. The effects of violent exertion in the production of this disease may be in some degree estimated by the fact ascertained by Bizot, that the popliteal artery, although very little invaded by atheroma, in proportion to some other arteries, is much more frequently the seat of aneurism than most other vessels.

There are not sufficient data to enable us to institute a comparison between the effects of different modes of employment. Some writers have hazarded generalizations on this point, but they evidently rest on very slender foundations.

But what are the causes which tend to induce the constitutional derangement that leads to atheromatous deposits? All circumstances which materially interfere with the process of digestion and assimilation *may* produce the atheromatous diathesis; they may, however, also produce scrofula, tuberculosis, diabetes mellitus, Bright's disease, &c. &c. The influence of impure air and bad food in producing Bright's disease, which in its nature is so nearly allied to atheromatous deposits, has indeed been seen in the experiments of Mr. Simon, brought forward by Dr. George Johnson, but the same has been shown by Dr. Lombard, of Geneva, in respect to scrofula. These circumstances may be taken to act as general, but not as special causes. In this sense spirit-drinking may possibly assist in inducing this state of the system.

Bizot has shown that sex has no influence; but that age, on the contrary, strongly predisposes to atheromatous deposits; that, in fact, they uniformly increase to extreme old age. Becquerel and Rodier have also shown, that after the age of forty, the quantity of cholesterine in the blood gradually increases. It is, however, a remarkable circumstance that atheromatous deposit is hardly ever found in the pulmonary artery and on the right side of the heart, except in those cases in which arterial blood is admitted to it by the imperfect closure of the foramen ovale. In what particular manner the vessels are thus influenced by the nature of the fluid which flows through them we know not.

The facts above stated are all that we at present know on this subject; but it is probable, from the rapid strides that organic chemistry has of late made, that more light may shortly be thrown on this, and on other points now buried in darkness.

CHAPTER VIII.

CASES OF THORACIC ANEURISM.

Case III.

Aneurism springing from the root of the aorta, and bursting into the pericardium.

A married woman, æt. 30, of very intemperate habits, had completely lost her appetite, and complained of severe gastrodynia and frequent vomiting. On being visited as a patient of the Birmingham Dispensary she was found with an anxious expression of countenance. The pulse was rather hurried. She had a cough, with slight expectoration, and tenderness in the epigastric region. She had occasionally palpitations of the heart.

The chest sounded clear on percussion. There were some slight cooing and bass-viol sounds heard in different parts. The *sounds of the heart were perfectly natural.* She improved considerably for some days, when, being excited, she suddenly dropped, and was found dead by Mr. Bromhall who was passing her residence at the time.

Inspection. There were signs of chronic inflammation in the coats of the stomach, and on the lining membrane of the bronchial tubes. The section of the liver had the appearance of nutmeg.

The pericardium was distended with blood, on removing which a tumour was seen nearly as large as a walnut, slightly shrivelled up. This was an aneurism which communicated with the aorta about one third of an inch above the valves at the back part of it, by an orifice as large as a sixpence, with rounded edges. The middle coat could be traced some distance into the sac; but the convexity of the sac was formed by the pericardium alone, and was lacerated in such a manner that the blood had escaped into the cavity of the pericardium. It was half filled with fibrinous layers, some of which were rather tough, and a small bit of which projected through the lacerated opening above mentioned.

It is seldom that an opportunity occurs of examining cases of this kind before death, the aneurism being generally so small at the time of its perforation as to have occasioned little or no inconvenience so as to induce the patient to seek advice. In this instance she applied for the relief of gastrodynia. On careful exploration no unusual sounds were detected in the precordial region. Even had a sound been engendered by the aneurismal pouch, it would undoubtedly have been mistaken for valvular murmur, on account of the proximity of the aneurism to the valves of the heart. It is therefore next to impossible to detect, during lifetime, the existence of aneurisms in this situation, where they seldom reach the size of a walnut. It is supposed by some that they are invariably formed by dilatation of all the coats, but in this case the tumour was formed at first by dilatation, and subsequently by the rupture of the inner and middle coats. This may also be inferred from the fibrinous layers found within the sac. A very similar case is related by Morgani, and in Dr. Smith's case, before alluded to, the greater part of the sac was formed by the pericardium alone.

Case IV.

Aneurism springing from the root of the aorta, and bursting into the pericardium.

A coach-harness forger, æt. 26, was taken suddenly ill at half-past seven in the morning. Mr. Meek was called in and found him in a state of collapse. His intellects were clear, and he complained of pain and a sense of tightness at the epigastrium. The pulse was almost imperceptible. His lips and the surface of his body were blanched and very cold, more especially the extremities.

The dullness of the precordial region was greatly extended and very marked. The pulmonary sound was clearly heard over both sides of the chest. The sounds of the heart were very distant, and the diastolic sound very indistinct; but there were no abnormal cardiac sounds. He sank in ten hours.

Inspection. There were traces of atheroma in the aorta. In one of the sinuses of Valsalva there was a dilatation of the size of a cherry, which had burst in the middle; and through this aperture a large quantity of blood had escaped into the pericardium, much of which was coagulated.

The absence of murmur in this case proves nothing; as, unless it were of a very marked character, it would probably have ceased when the action of the heart became oppressed by effusion of blood into the pericardium.

I have seen three cases very similar to this; but, as the patients had not complained, and consequently were not examined during lifetime, a detailed account of the post-mortem appearances would be useless. I have seen also the diseased parts in two other cases which occurred in the practice of Mr. Simons and of Mr. Wilkinson.

Case V.

Aneurism springing from the concavity of the arch of the aorta, and bursting into the pericardium.

A wheelwright, æt. 40, of intemperate habits and addicted to spirit-drinking, had an attack of acute rheumatism three years since; and from that time had suffered occasionally from flying pains of a rheumatic character; and for some weeks he had felt some pain across his chest. He was attended by Mr. Meek who, on a careful examination of the chest, failed to detect any abnormal sound.

The evening before his death he boasted of being quite well and free from pain. He had bread and cheese and a pint of beer for supper, and retired to bed at half-past ten. A little before midnight he got out of bed, but did not complain. About two o'clock in the morning his wife awoke and spoke to him. Finding he did not answer, she raised him up in bed, when he smiled at his child, gave a gasp, and died.

Inspection. The pericardium was filled with liquid and coagulated blood. The heart was very fatty. The aorta was studded with atheroma, which had extensively invaded its middle coat. In the concavity of the arch of the aorta was a hole with rounded edges of the size of a fourpenny-piece, that opened into a pouch of the size of a hen's egg, which passed downwards, and the back of which closely adhered to the pericardium. The middle coat of the aorta only passed for a short distance into it. It crossed the bifurcation of the pulmonary artery. Having been opened immediately on its removal from the body, the aperture through which the blood passed into the pericardium could not be discovered, having probably been very small, and having been cut directly through with the scalpel.

There was not a single sign of aneurism present in this case; indeed it is difficult to see how there could be. For although the sac sprang from the concavity of the arch of the aorta outside of the pericardium, yet its adhesion to this membrane caused it to burst into its cavity without attaining any large size. As far as relates therefore to diagnosis it may be classed with aneurism arising from the root of the aorta within the pericardium.

The last two cases were brought under my notice by Mr. Meek, who was also kind enough to present to me the diseased parts, which have been placed in the Museum of King's College, London.

Case VI.

Aneurism of the arch of the aorta communicating with the trachea.

A tradesman, æt. 38, had suffered some months from hoarseness and partial loss of voice. He was constantly troubled with a cough, and had several attacks of hemoptysis, in which black coagulated blood was generally brought up, mixed with a little of a bright red colour. He constantly felt a constriction of his throat, and his respiration was accompanied by a slight whistling. The trachea seemed to recede backwards from the sternum, but was not turned on either side. No trace of any anormal sound could be detected over any part of the chest.

In this state, he was one day suddenly seized with symptoms of acute laryngitis. Mercury was rapidly administered, and preparations were made by Mr. Lawrence, under my advice, to perform tracheotomy, had not relief been obtained. He was, however, quickly salivated, and recovered, but the cause of these attacks still remained concealed from view.

Some months after this, he was seized with symptoms of severe bronchitis; on this occasion he was seen by Mr. S. F. Palmer, who was kind enough to send me the particulars of the attack, and of the post-mortem appearances, and to show me the diseased parts. From his account, it appears that previous to death the patient was expectorating a thin sanious fluid, that whilst conversing he was suddenly seized with symptoms of suffocation, gasped eagerly for breath, attempted to thrust his fingers down his throat, as if to remove some obstacle to respiration, sank back upon his bed, and died.

Inspection. There was much atheromatous and some calcareous deposit in the aorta. The arch was dilated uniformly into a pouch, at the back of which was an oval opening, an inch long and half an inch wide, with smooth rounded edges, leading into a sac which would have held a chesnut. This sac was bounded at the sides by the arterial coats, and at the back by the trachea, to which it closely adhered, and with which it communicated by a small aperture with smooth and rounded edges, capable of admitting a crow-quill. It was filled with concentric layers of fibrin, the most external of which were tough and light coloured. This sac pressed on the trachea, and very much diminished its calibre.

"The larynx presented evident signs of chronic or repeated inflammation, the submucous tissue being evidently thickened. There were signs of inflammatory action in the lining membrane of the trachea."

There was not, from first to last, a single sign that could be taken to indicate the existence of aneurism. Neither the dilatation of the arch of the aorta, nor the pouch, were sufficiently great to interfere with any other part than the trachea. The whistling respiration might have been produced by chronic laryngitis; and both this and the hemoptysis might have been referred to tuberculization of the lungs; but the absence of all physical and general signs of this disease excluded this cause. To me the symptoms were inexplicable; but it is possible that during the attacks of hemoptysis some fragments of fibrinous clots might have been expectorated, which, had they been seen, would have raised a suspicion of the nature of the affection; as in Cases IX and XXIII.

Case VII.

Aneurismal pouch springing from the arch of the aorta, and compressing the right bronchus.

A married woman, æt. 50, had been subject to asthma for some years, and to occasional paroxysms of urgent dyspnœa. In one of these she was admitted into the Birmingham General Hospital, and died before she could be carefully examined.

Inspection. The right lung was much smaller than the left, and contained but little air. A few tubercles were scattered through its apex, but were not found in any other part of the body.

An aneurism, of the size of an orange, was seen springing from the right side of the ascending aorta, and compressing the right bronchus and the vessels of the corresponding lung. It communicated with the aorta by a circular aperture of an inch and a half diameter. The middle coat of the aorta was traced for a short distance into the aneurismal pouch, which was lined with fibrinous layers.

Had it been possible carefully to examine the chest in this case, it is probable that the compression of the right bronchus would have been discovered by the absence of respiratory sound on the right side of the chest, in which case, had an aneurismal murmur been heard in that spot alone, the nature of the affection would have been made out, as in Case xi. But no physical signs of less value than this would have sufficed, as the bronchus might have been compressed by a solid tumour. This case was brought under my notice by Mr. W. C. Freer, house-surgeon of the hospital.

Case VIII.

Mixed aneurism of the thoracic aorta communicating with the left lung.

A boatman, æt. 30, eight months ago was attacked with a dry cough, and pains flying about his chest, more especially his left shoulder; two months ago his cough became violent and convulsive, and he expectorated a viscous fluid, often streaked with blood.

On his admission into the Birmingham General Hospital, his face was tumid and his lips purple. He preferred a sitting to a recumbent posture, as the latter, more especially when he lay on his right side, produced severe headaches. He had great dyspnœa. His cough came on in fits, and resembled the barking of a dog. The expectoration was thick, scanty, and clear. His voice was hoarse. He complained of a dull, aching pain in his chest, shooting backwards. He perspired greatly during the night. His pulse was 72; soft.

A shade of dullness was observed at the left of the top of the sternum. The pulmonary sound was accompanied by cooing and hissing sounds. The impulse of the heart was weak, and the sounds feeble but distinct. Soon after this he took cold, and his bronchitis became much aggravated; this, however, was relieved, and he left the hospital.

A week after this, having exerted himself, he expectorated half a pint of blood, and in another fortnight a pint. He was readmitted, labouring under troublesome cough and dyspnœa. He had lost flesh, and his lips and cheeks appeared to be slightly congested. He complained of violent pain in the left temple. The dyspnœa and cough were both increased by exertion, and also by his assuming the recumbent position. The voice was hoarse and unequal, the cough barking, sometimes followed by palpitations. The expectoration was aqueous and frothy. He had intermittent pain darting from the right scapula through to the front of the chest. The trachea seemed pushed a little towards the right side. On a level with the nipple, the left side measured a little less than the right. A pulsation, but no purring thrill was felt between the cartilages of the first and second left ribs, close to the sternum. In that spot there was dullness on percussion, and a tracheal whistling was heard very close to the ear. The sounds of the heart were rather extended. Soon after this, he stated that he experienced a difficulty in swallowing solids, owing, as he supposed, to some obstacle at the bottom of the neck; but that when one morsel had passed, he could swallow his meal with ease. One day he perceived a tickling in the throat, which was followed by the expectoration of half a pint of blood, at first solid and black, then red and fluid. At this time a greater dullness than usual was perceived on percussing the supra-spinal region of the left scapula. The tracheal whistling was very strongly marked over the dull spot to the left of the top of the sternum, where the sounds of the heart were very audible, the diastolic being louder than the systolic, which was feeble. After another expectoration of two pints of blood, the dullness of sound and pulsation to the left of the top of the sternum were much less marked, but reappeared on his raising himself up. Dullness was more marked behind, where a fine, moist rattle was heard. About this time, it was thought that the pulmonary sound on the left side of the chest was jerking, as if interrupted for a moment every now and then. The diastolic sound of the heart became raspish, between the second and third left costal cartilages, and slightly so above the left nipple; this was never observed before. Great oppression of the chest succeeded, and he died from hemoptysis.

Inspection. All dullness had disappeared from the front of the chest. The lungs did not collapse, being retained in their position by adhesions, chiefly at their summits and on the left side.

On dissecting down towards the vertebral column, a tumour was perceived, and found to be a dilatation of the descending aorta; which commenced immediately below the origin of the left subclavian artery, on a level with the upper edge of the third dorsal vertebra, and extended to the upper edge of the sixth. It adhered closely to the vertebræ included between these two points, from a little to the right of the mesian line, to the bases of the left articular processes. On its right side lay the trachea and œsophagus, a little pushed out of their proper places. Its anterior surface was crossed by the left bronchus, and by the recurrent nerve; the former of which was much compressed, and the latter was stretched, but not altered in structure. The sac also adhered closely to the left lung. Its cavity might have contained a moderately-sized pear. The lining membrane was much thickened and puckered up with atheroma. The middle coat was traceable throughout the pouch, with the exception of the place where the bodies of the vertebræ were involved, and also a spot of the size of a half-crown-piece, which was an aperture opening into a cavity in the back part of the substance of the apex of the left lung as large as a chesnut, which was lined by a smooth membrane in some parts; in others the lining was roughened by irregular tufts of coagulated fibrin, and was surrounded by a dense uncrepitating substance. The bodies of the vertebræ implicated were deeply excavated, leaving the cartilages between them projecting into the pouch; they were, however, covered by a smooth membrane which appeared in all respects as if it were a continuation of the lining membrane of the aorta. Below the dilatation the coats of the aorta were much thickened and diseased. The heart was very small and pale. On cutting into the left lung a great portion of its apex around the cavity above mentioned was carnified, and contained some small groups of gray granulations. Immediately around the cavity was a mass of laminated fibrin, one inch thick in the middle, but tapering off on all sides towards the edges of the opening into the aorta.

Several bronchial tubes were traced down from the trachea, and were found to open on the fibrinous clot, their lining membrane being stained of a purple red colour as they approached it. In one spot a secondary branch was dilated into the cavity of the size and figure of an almond without its shell.

The abdominal viscera were healthy.

There was abundant evidence of the existence of a tumour compressing the trachea, œsophagus, and recurrent nerve, and in contact with the aorta; and it was further probable that it was connected with the left lung and the spine. Aneurism was of course suspected but not predicted with confidence. Further consideration has convinced me that the diminution of the extent of dullness on percussion after copious hemoptysis, and which was observed more than once, could only be accounted for on the supposition that the tumour was an aneurism imbedded in the left lung.

The above case was frequently examined in company with the house-surgeon, Mr. Alfred Baker, by permission of the physician, Dr. Evans, and with his consent this abstract of the case, abridged from Mr. Baker's notes, has been published.

Case IX.

Sacculated aneurism of the descending aorta—Death from rupture into the trachea.

A pig-killer and night-constable, æt. 46, had been for some months addicted to spirit-drinking. Seven months since he had a pain in the lower part of the front of the chest. Thirteen weeks ago, having one night drank a great deal of raw brandy, towards morning he was seized with dyspnœa and a partial loss of voice, which has never regained its power, but has remained little more than a whisper. Soon after this, one night whilst on duty he suddenly fell down, and on coming to himself felt very giddy and his limbs were numbed, from which he recovered in the course of the next day, but has ever since felt a severe pain between the shoulder-blades shooting up and down the back. A fortnight since he expectorated nearly half a pint of clotted, dark, mottled-coloured blood, which (to use his wife's words) looked as if it came from an old ulcer. The day before he was seen he expectorated a little more of a similar kind.

When visited as a patient of the Birmingham General Dispensary he complained of great dyspnœa and severe pain between his shoulders. The pulse was natural.

The chest sounded well on percussion. Over the right side of the chest the pulmonary sound was pure but very intense. Not a trace of it could be heard on the left side. The action and sounds of the heart were natural. When the patient spoke, a slight vibration was felt by the hand placed on the right side of the chest, but none on the left. The pain was relieved by the application of leeches.

He was suddenly choked eight days after he was visited at home.

Inspection. The lungs were quite healthy, as also the heart, except that there was opacity of the lining membrane of the left auricle and some patches of atheroma under it. The inside of the aorta was covered with such patches.

A tumour rather less than a hen's egg was found firmly attached to the commencement of the descending aorta, and also to the trachea just above its bifurcation, and to the left bronchus, which was compressed and flattened by downward and backward pressure. On cutting into it, its walls were found to be thin, and it was seen to be a sacculated aneurism of the aorta, with which it communicated by an opening commencing close to the transverse portion of the arch, and extending one inch downwards and half an inch in width. Its shape was thus oval. It was filled with layers of solid, tough, discoloured fibrin, and for some distance around the opening into the aorta was a smooth lining membrane apparently continuous with that of the aorta, but the middle coat seemed to stop abruptly. It had ulcerated, and perforation had taken place into the trachea at the point of adhesion just above its bifurcation. Through this opening a piece of firm old coagulum had passed into the trachea. A slight quantity of recently coagulated blood was found in the left bronchus and trachea.

The only physical signs observed in this case were loss of voice, and a complete absence of respiratory sound, with persistence of clearness on percussion over the left side, evidently caused by the pressure of some small tumour on the left bronchus, which did not compress the trachea to any sensible extent. The blood which the patient had expectorated the day before he was visited having been preserved was found to contain fragments of tough discoloured fibrin. The tumour was, therefore, suspected to be a sacculated aneurism of the descending aorta, which, as in Case VI, communicated with the bronchus or trachea. Unfortunately the connexion between the tumour and the recurrent nerve was not investigated. The membrane which lined the sac for a certain distance was, doubtless, the fine false membrane described by Bizot.

Case X.

Aneurismal pouch springing from the upper and right portion of the arch of the aorta, and compressing the trachea and the upper branch of the right bronchus.

The Adjutant of a dragoon regiment, æt. 45, of athletic frame, had been accustomed to take a great deal of violent exercise. When a young man he had drunk hard, but of late years had been very temperate. For three years he had suffered more or less from dyspnœa and cough. About a year since these symptoms became aggravated, and a severe pain came on between his shoulders, with a sense of tightness across his chest; and his cough invariably became urgent on lying down. Having been much fatigued and chilled by a long march on a very cold wet day, and being also much excited, his eyes were observed almost to start from his head, and his face became very livid. A pain in the right side succeeded, and he was attended by Mr. Hodgson, in consultation with the surgeon of the regiment, when symptoms of slight pleuro-pneumonia at the lower part of the right side were observed. Dyspnœa, cough, and constriction of the chest remained, however, after the acute attack had subsided. Seen in consultation with Dr. Pilkington, he stated that he had not been able to lie down for fifteen weeks. He had a harassing cough with a slight amount of frothy expectoration, and complained of great tightness of the chest, and severe pain shooting from under the sternum backwards and down each arm. He breathed freely when considerably inclined forwards, and in that position he could run about the room without distress; but the moment he assumed either the erect or recumbent position, he was threatened with suffocation. He was cheerful, and stated that his appetite was good and his bowels regular. His pulse was feeble, but natural, his tongue slightly coated. No pulsation or thrill could be felt in any part of the chest. The impulse of the heart was feeble.

The chest sounded rather dull over a small spot to the right of the upper angle of the sternum, and also near the bottom of the posterior portion of the right side. The pulmonary sound was rather coarse, and with it were traces of small crackling and bass-viol sounds. When he raised himself upright the respiratory sound totally ceased over the upper third of the right side of the thorax, and a whistling sound was heard at a distance from the patient. The sounds of the heart were natural, but were heard rather louder to the right of the upper corner of the sternum than in the precordial region. He was much relieved by belladonna frictions over the chest; but in a few weeks' time he was seized suddenly with a violent paroxysm of coughing, having a few days previously taken cold as he thought. This continued for three hours incessantly, when he died.

Inspection. A tumour of the size of a large orange was found lying under the upper part of the sternum and to the right of it, but not adhering to it; and seemed to have been nearly covered by the left lung when inflated. It adhered closely to the trachea immediately above its bifurcation, and for three inches upwards, and considerably compressed and even flattened it. It also over-lapped the right bronchus, more especially a large upper branch, which was given off almost immediately below the bifurcation of the trachea. This tumour was found to be an aneurismal pouch, partly formed by the dilatation of the upper right angle of the arch of the aorta and the orifice of the arteria innominata, and partly by distension of the cellular coat. It was about half filled with layers of old-formed, discoloured, fibrinous coagula. The heart was of moderate size, and of healthy appearance. Both lungs, but more especially the right, were much engorged. At the bottom of the right lung, towards its posterior part, was a mass of gray tough induration, about the size of an orange.

The preparation is in the Museum of King's College, London.

There were not in this case any of the usual signs which are supposed to indicate aneurisms. There was no unusual pulsation or thrill, nor any anormal sound whatever; except the whistling respiratory sound which denoted compression of the trachea. The tumour, in fact, had hardly reached the surface of the chest, so that no thrill would be felt, nor any pulsation, except it were propagated up the trachea. Had the left ventricle been hypertrophied, it might have produced a strong current through the pouch, and thus have given rise to murmur, which in its natural state it could not be expected to do. But in the absence of all ordinary signs, there was one found which clearly indicated the nature of the affection. For it was not only evident that the trachea was permanently compressed by some tumour, but that this tumour extended considerably to the right, and was so moveable that in a certain position it fell upon the upper division of the right bronchus, and on that alone. Had the tumour consisted of diseased glands, it would not have been thus moveable, but have been one solid mass, as in Case XXVII. An aneurism springing from the right of the arch of the aorta therefore alone remained as the possible cause of this peculiar sign. This, therefore, was the diagnosis given.

Case XI.

Dilated aneurism of the arch of the aorta, compressing the superior vena cava.

A bargeman, æt. 40, of a spare habit of body, and addicted to hard drinking, had been asthmatic, and had at different times felt palpitation of the heart for the last ten years. He was intoxicated for a week, during which time his head and neck swelled, but more particularly during the last night of the week, and he awoke early in the morning with great stiffness and pain in his throat, and in the epigastric region. His head, neck, and arms were found to be amazingly swelled, and his face was almost black.

When visited as a patient of the Birmingham General Dispensary, his head, neck, arms, and the upper part of his chest were greatly swelled, of a purple colour, and pitted on pressure. His eyes were injected and watery. He suffered much from dyspnœa. The pulse was feeble and resilient. The action of the heart was strong and heaving.

The natural sounds of the heart were distinctly heard in the precordial region. The chest sounded rather dull on percussion throughout, owing to the œdema of its walls; but no one part sounded duller than another. The breathing was rather stridulous, even when heard at some little distance off. The respiratory sound was natural, but slightly modified by the stridulous breathing. About four inches from the top of the sternum, and one inch to the right of it, an extremely hollow and rather prolonged systolic rasp-sound was heard, and was quickly lost on the ear receding from this point. Ineffectual attempts were made to bleed him in both arms. Erysipelas came on in each arm around the incision. In a few days the œdema of the head, neck, and arms was much diminished, but the erysipelas progressed, and he died.

Inspection. The tumefaction of the head, neck, and arms had nearly disappeared. The ravages of the erysipelas were confined to the subcutaneous cellular tissue. The lungs were healthy. The heart was firm, red, and hypertrophied, being half as large again as the fist; but the size of its cavities was but little increased, nor were its valves diseased.

A tumour occupied the upper part of the mediastinum, and projected to the right of the sternum. This was the arch of the aorta dilated into a pouch capable of containing a small orange. The arteria innominata was also slightly dilated at its origin, like a funnel. The coats of the aorta could be traced throughout every part of the pouch but one, and this was a small spot at the back of it, where a tough old fibrinous clot adhered to a bronchial gland without the intervention of any of the arterial coats. A more recently formed clot adhered to the summit of the pouch, and sent off branches into the arteria innominata and left carotid artery. This pouch had overlapped the superior vena cava, which adhered closely to its posterior surface. By this means the vein was flattened, and contained a firm, flat, and somewhat discoloured clot. The left subclavian vein was twisted, its insertion into the vena cava being drawn down under the pouch.

The existence of a tumour compressing the superior vena cava and the trachea was at once apparent. A hollow rasp-sound being there found confined to the spot where such a tumour might be supposed to lie, sufficed to reveal the nature of the tumour, and to determine its being an aneurism of the arch of the aorta. The hollow character of this sound rendered it probable that the aneurism was the result of dilatation, a supposition which was to a certain extent confirmed by the resilient character of the pulse.

Case XII.

Aneurism of the thoracic aorta.

A female servant, æt. 25, received a violent shock from a fright eleven months ago, since which time she has been very nervous and hysterical. She has, in the course of this period, had several attacks, which were chiefly characterized by severe pain in the chest, dyspnœa, palpitation, and on two occasions she lost her voice for some days. After one attack her right hand and arm swelled, and became of a purple colour, which lasted ten days or a fortnight.

Having been admitted into the Birmingham General Hospital, her face was seen to be turgid, and she complained of occasional giddiness and tinnitus aurium, and throbbing of the vessels of the neck, of pain at the precordial region, shooting towards the left shoulder and down the arm, and of constant severe pain between the shoulder-blades. She was unable to lie down without experiencing the sensation of suffocation, and violent palpitations appeared on the slightest exertion or mental emotion.

The impulse of the heart was strong and jerking. There was violent pulsation in the carotid arteries; a strong purring thrill was felt on pushing the finger down behind the summit of the sternum, and also between the third and fourth right costal cartilages. This spot sounded dull on percussion. The precordial dullness was lower than usual. There was a little coarseness in the respiratory sound generally. There was a soft systolic bellows-sound heard mostly at the situation of the apex of the heart, and also behind the left side, accompanied by the diastolic sound, which was rather less clear than usual. At the point to the right of the sternum, where the dullness and purring thrill were perceived, there was a double saw-sound, which became single on approaching the right clavicle, and was slightly prolonged into the carotid artery.

She remained in the hospital some time, during which she had an attack of acute articular rheumatism, after which the pulsations, thrill, and murmurs were much more feeble. She was relieved, and has not been since heard of.

Although there was probably in this case some disease of the valves of the left side of the heart, and much hysteria, yet the evidences of aneurism were also clear and unequivocal. At the spot where there were dullness and a thrill, there was a diastolic sound, which was not heard at all at the heart. The aorta was dilated at its arch, as could be distinctly felt with the finger.

Case XIII.

Mixed aneurism springing from the concavity of the arch of the aorta.

A stonemason, æt. 48, applied for advice. He complained of pains flying about him, particularly in the chest, and of dyspnœa. Signs of slight bronchitis were detected in the chest, but nothing beyond this. He was relieved, and applied again in six months. He then complained of a troublesome cough, and occasional severe fits of palpitation. The respiratory sound was natural, but was absent to the left of the upper part of the sternum. The impulse of the heart was strong. The pulse at the right wrist was decidedly more feeble and much smaller than that at the left wrist.

Between the second and third left costal cartilages, a loud hollow systolic rasp-sound was heard, the intensity of which rapidly decreased in all directions, but still could be heard at the precordial region. Over this spot there was slight dullness of sound on percussion, and absence of respiratory sound, as has been stated, and it was thought there was slight fulness of the space between the cartilages. He was relieved by small bleedings, &c. &c.

Six months after this he was visited, and found in bed. He was thin, and was excessively troubled with cough, accompanied by clear expectoration, and by pain under the sternum, shooting both backwards and down the right arm, and great constriction across the chest. The pulse in the right wrist was almost imperceptible. The action of the heart was strong and heaving. Between the cartilages of the second and third left ribs, close to the sternum, a dull sound was given out on percussion, and an elevation as large as a half-crown was perceived. A pulsation not very liquid, but no purring thrill, was felt over this spot. No pulsation was felt over the top of the sternum, or over either clavicle. The respiratory sound was accompanied by a little cooing in spots. A short raspish systolic sound was heard over the left nipple, increasing in intensity as the ear approached the elevation between the second and third cartilages of the left side, where it was louder and more hollow. Mild sedatives were ordered, and cold applications to the elevated spot, but he did not bear this well, his pain being much increased by it. One day he felt something burst under his breast-bone, and the blood seemed to rush up to his head and ears, after which the pain and constriction of the chest and dyspnœa became much less. In another six months the pulse in the right wrist was still extremely small, and his right arm had for some time felt rather benumbed. This he attributed to its being much out of bed, as he could only lie on his left arm, with his right arm crossed over him, without urgent dyspnœa. He had also twitchings in his right arm, and thought his hearing was impaired.

Between the cartilages of the fourth and sixth ribs on the left side there was an elevated cone, with a base as large as a shilling, a slight elevation spreading for an inch or two around it. There was dullness for some inches around this spot, and pulsations were felt all over it, not very liquid. A systolic rasp-sound was heard very loud over the centre, decreasing as the ear receded from it, but still heard above the left nipple. Behind, the respiratory sound was accompanied by much muco-crepitant rattle, and in front it was whistling, as if the trachea was compressed. The expectoration was usually clear and tenacious, but he had once or twice spat a little blood.

He sank gradually; greatly emaciated.

Inspection. The external prominence had nearly subsided. On opening the chest, a large tumour was seen lying under the sternum, and projecting into the left side more than an inch beyond the point of the union of the second, third, and fourth ribs with their respective cartilages, and lying on the base of the heart, which was thus slightly pushed downwards. The left lung was compressed by it, and, being cut into, was found carnified of a dark slate colour. The heart was large, firm, and red, but its valves, though a little thickened, acted quite well. The aorta, from within half an inch of the semi-lunar valves up to the origin of the left subclavian artery, was found to communicate with an enormous pouch, which thus involved the greater part of the concavity of the arch. On careful dissection, it appeared that the middle coat of the aorta did not cover more space than it would have done had it been laid open in a healthy undilated state. The natural channel of the aorta was rendered rugged and uneven by the deposit of atheroma, and by semi-cartilaginous ridges. The greater part of the sac to the left was formed of the outer coats of the vessel, and near the ribs these had apparently given way. At this spot a very large, thick, yellowish-brown, fibrinous clot adhered; its base was as large as a shilling, and it extended more than an inch into the sac. It had abrupt edges, and was covered with a smooth membrane, which was reflected from it on the walls of the sac, where it soon became rough and granulated, as if coagula had been detached from it. To the right of the sternum was a similar clot, partly detached from that bone, and hanging loosely into the sac. The origin of the right subclavian artery was completely plugged up by firm coagulum.

On the first visit of this patient it is probable that only a slight examination of the chest was made. On his second visit, the state of the pulse in the right wrist led to a very careful exploration of the chest, when signs of solid matter were found to the left of the upper part of the sternum, and as in the same spot a very hollow rasp-sound was heard, much louder than at the precordial region, it was concluded that the arch of the aorta was dilated, and perhaps also ruptured at its left angle; and the mouth of the arteria innominata partially closed by atheroma, coagulum, or horny patches. The quality of the rasp-sound was the same as that heard in Cases XI, XVIII, and XX, and was more hollow than I have ever heard when it was formed at the orifices of the heart. In a short time the nature of the affection was made manifest, by the appearance of pulsation. After the sensation of bursting, the aneurism pointed in a spot two or three inches lower than where it first appeared, owing to the rupture of the coats, or the spreading of the sac downwards.

Case XIV.

Sacculated aneurism springing from the ascending portion of the arch of the aorta, and bursting externally.

A working jeweller consulted Mr. Baynham, and stated that eight months previously he had felt pains in the loins, and that about five months ago he was "much put out," when he felt a sudden snapping under his breast-bone, accompanied by pain, which had continued ever since, striking from under the right nipple to the back between the shoulders. He also stated that he could not lie on his left side without greatly increasing the pain in his chest.

When visited in consultation with Mr. Baynham, there was a violent throbbing of all the arteries, the pulse being sharp, quick, and rather full; and a strong heaving impulse at the precordial region. A purring thrill was felt to the right of the upper third of the sternum. Over this spot there was a dull sound on percussion. A bellows-sound was heard following as close as possible upon the systolic sound, all over the chest. Ten days after this, he stated that he had latterly felt violent pains in the back, between the spine and the angle of the right scapula. Pulsation was felt where the dullness and thrill existed.

The notes of the case are incomplete, but, nine months after he was first seen, the upper half of the front of the right side of the chest projected forwards, over which part very liquid pulsations existed. Over the whole of the swelling a double rasp-sound was heard. He could not support cold applications.

In three months more the projection of the upper half of the front of the right side of the chest had almost entirely receded; but a conical tumour pointed close to the middle of the sternum, a little to the right of the mesian line. Over this spot there was violent pain. He was still unable to bear cold applications, owing to the pain they occasioned. The legs became œdematous. The tumour rapidly increased in size, and its apex became red and shining; blood began to ooze from it, and at length it burst externally, and death ensued.

Inspection. The upper two thirds of the right half of the sternum, as well as the ribs and cartilages on that side, from the first to the fourth inclusive, were found adherent to a large tumour, which occupied the upper half of the right side of the chest, and had compressed the upper lobe of the lung into a very small space. The heart was larger than natural, from hypertrophy of its walls, chiefly those of the left ventricle. It was red and firm. The aortic valves were thickened, but acted freely. The internal surface of the aorta was thickened, and studded with many patches of atheroma and horny plates. At the convexity of the ascending portion of the arch, from within an inch of the semi-lunar valves to the origin of the arteria innominata, was a circular opening as large as a crown-piece, which communicated with an enormous pouch, which constituted the tumour above mentioned, and which was the size of a largish melon, and could contain more than a pint of fluid. Its lining membrane seemed intimately blended with that of the aorta, but the middle coat of the latter terminated about two inches from the aorta, all round the aperture. In this sac there was no coagulum. A portion of the walls of this pouch consisted of the right half of the upper two thirds of the sternum and of the costal cartilages, and a part of the ribs which were inserted in it. The greater part of this portion of the sternum was devoid of periosteum, and was much thinned by erosion. The cartilage of the first rib was nearly, and those of the second and third ribs were entirely separated from the sternum, and from their intercostal muscles. Between the second and fourth cartilages was an aperture of the size of a half-crown piece, which led into another sac external to the sternum, eight inches in circumference and an inch and a half in height. In it were some old, reddish coagula, and some recently clotted blood, and it was partially lined within by a smooth membrane. The integuments were very thin, and had given way at the spot where the dark purple appearance had been observed during lifetime.

The history of this case, coupled with the post-mortem appearances, lead to the belief that the disease commenced by dilatation of the ascending aorta, and was followed by rupture in a fit of passion. When he came under my notice there were evidences of hypertrophy of the heart, and there was a strong presumption in favour of the existence of a sacculated or mixed aneurism approaching the surface of the chest to the right of the upper third of the sternum. This presumption was based on the existence of a purring thrill and dullness on percussion over this spot, on the extent to which the rasp-murmur was heard, and on the fact of its not being exactly synchronous with the impulse of the heart. This was soon converted into certainty by the appearance of a liquid pulsation. The sheath and the pleura seem at length to have given way, and the blood passing forward between the ribs, was bounded for a time by the integuments of the chest alone. The murmur was probably formed in the roughened aorta.

Case XV.

Aneurism of the pulmonary artery—Constriction of the aorta, &c. &c.

A thin pale girl, æt. 19, a screw-wormer, was a patient of the Birmingham General Dispensary, under the care of Dr. Fletcher, and by his kindness was submitted to my examination. She complained of violent pain in the head, and of some pain in the left side of the chest, great dyspnœa, and troublesome cough.

Immediately under the left of the sternum, between the second and third ribs, there was dullness on percussion over a space measuring nearly four square inches. Over this spot a very liquid pulsation was felt, accompanied by a purring thrill so rough and so close to the surface, that it seemed almost to grate on the fingers. At the same place a loud hollow rasp-sound was heard with the systole of the heart, and also for a moment at the commencement of the diastole, as if the blood was receding.

This case has been detailed at length by Dr. Fletcher, in the 25th vol. of the Medico-Chirurgical Transactions, from which it appears that the patient lived a year after this examination, and, after repeated attacks of bronchitis and pneumonia, died rather suddenly.

Inspection. The pulmonary artery was found dilated into a pouch, the interior circumference of which measured nearly six inches, and which pointed principally in the anterior direction, where the internal and middle coats were wanting, and where a fibrinous clot was found. One of the pulmonary valves was contracted. The aorta was first a little dilated and then contracted, particularly where it was joined by the ductus arteriosus. There was also a permanent communication between the two ventricles.

Dr. Fletcher's description is accompanied by a drawing, and is well worthy of perusal; and he has been kind enough to show me a beautiful preparation of the morbid parts.

The extreme proximity of the thrill and rasp-sound to that part of the surface of the chest which covers the pulmonary artery, led to the belief not only that this sound was engendered in that vessel, but that it must have been brought up close to the surface by its dilatation. Doubtless a part of the sound was engendered at the constricted portion of the aorta, as in Cases XXIX and XXX.

CASE XVI.

Aneurismal pouch springing from the right side of the ascending aorta—Right pleuritic effusion.

A widow, æt. 44, had experienced palpitations at the heart for three years, and had been subject to occasional attacks of rheumatism. Six months since she was suddenly seized with acute pain in the right side; this was succeeded by great dyspnœa and flying pains in her shoulders, and fixed pains in two of her fingers, which were red and swollen.

Seen in consultation with Mr. Meek, she was found rather emaciated, with some œdema of the extremities. Her pulse was feeble, and her bowels constipated. She had a slight cough, with clear frothy expectoration. She suffered from severe palpitations, and occasional paroxysms of most urgent dyspnœa, accompanied by a great sense of terror; so that she would hardly allow any one to approach her at such times.

Below the nipple, the right side of the chest measured two inches more than the left side, and on a line with the axilla one inch more. To the right of the upper part of the sternum a pulsation was felt much stronger than in the precordial region, and also, though faintly, all over the right side. Excepting immediately under the clavicle, the whole of the right side of the chest sounded as dull as possible. On the left side the precordial dullness was rather more extended than usual. For two inches below the right clavicle, and nearly half way down the back of the right side, a strongly marked blowing tracheal sound was heard, and which appeared very distant, as if it were propagated from the bronchus of the opposite side; below this point no pulmonary sound could be detected. On the left side the respiratory sound was rather coarse, with some traces of muco-crepitant rattle. Over the precordial region a double bellows-sound was heard, which became louder and more hollow and coarse towards the right of the upper part of the sternum, where it amounted to a rasp-sound. She lived only a few days after this examination, and died in one of her paroxysms of dyspnœa.

Inspection. The right side of the chest contained more than a gallon of serum. The right lung was compressed against the spine, and devoid of air. Several membranous bands passed from this lung to the sides of the thorax, and to the diaphragm. The left lung was very voluminous, and adhered closely to the chest. The heart was very large. Under the upper half of the sternum was seen a round tumour of the size of a large orange, which projected two inches to the right of the sternum, and adhered to that bone over a space as large as a shilling. It lay over the right bronchus and descending vena cava, but was not adherent to either. It was found to be an aneurismal pouch springing from the right of the ascending aorta.

The true nature of the affection was in this case disguised by the pleuritic effusion in the right side of the chest. When I saw the patient the chest had not been measured, and I certainly mistook the impulse of the aneurism for that of the heart, which I thought had passed over to the right side, as in Cases LXXXVI, LXXXVII, and LXXXVIII. It was impossible to examine the case thoroughly, owing to the excited state of the poor woman. The murmur might have been produced at the heart; the dullness on percussion extended over the whole side, and the pulsation, modified by the fluid in the pleura, more resembled the impulse of the heart than an aneurismal pulsation. From whatever cause, the nature of the affection was not detected.

This and the following case were kindly brought under my notice by Mr. Meek, who also furnished the notes of the inspection, and presented me with the morbid parts, which are now in the Museum of King's College, London.

Case XVII.

Aneurism of the arch of the aorta.

A widow, æt. 42, took cold from exposure four years ago, since which time she has had a dry cough, and her breath has gradually become shorter. Two years since she fretted very much in consequence of the loss of an only child. During the last nine months she has constantly experienced a little pain, which seemed to shoot from the left of the upper part of the sternum backwards through her breast, and has been much increased by coughing, and which has been rendered excruciating by pumping or any other violent exertion. During the last six months she has had frequent palpitations, which have been always aggravated by exertion or moral emotion.

The pulse was small, particularly when compared with the impulse of the heart, which was very strong three inches below the left nipple. No pulsation or thrill could be felt over the sternum or clavicles.

Immediately to the left of the upper third of the sternum a prominence was observed, rising gradually to the height of about half an inch, and covering a space about twice the size of a crown-piece. Over this spot there was a faint pulsation felt, which was not at all liquid. A coarse double murmur was also heard in the same place. At the precordial region both the natural sounds of the heart were heard, unaccompanied by any murmur. The respiratory sound was natural. Leeches and belladonna frictions were ordered to the region of the heart, a mild anodyne mixture was prescribed, and a bladder, containing a refrigerating mixture, was directed to be kept constantly on the tumour. In a few weeks' time the tumour had sensibly diminished, and the pain and palpitations were much less severe. She has not been since heard of.

The aneurismal pouch, in all probability, already contained fibrinous clots when the patient came under examination. The existence of a double murmur confined to the spot where a pulsating tumour was seen, afforded conclusive evidence of the nature of the tumour, and clearly proved it to be a sacculated aneurism of the aorta.

Case XVIII.

Dilated aneurism of the arch of the aorta, communicating with the left lung, and bursting into the left pleural cavity.

A Thames waterman, æt. 32, accustomed to drink hard, four months since had an apoplectic fit, for which he had been bled, cupped, and blistered with relief, but was left with impaired motory power of the left side of the body, and a constant dull aching pain on the left side of the head.

Admitted into St. Thomas's Hospital, under the care of Dr. Elliotson, he was found labouring under considerable dyspnœa, which was much increased on exertion. His appetite entirely failed him, and he could not sleep. The bowels were costive. No pulse could be felt in either arm. He was troubled with violent coughing on attempting to lie flat.

The chest sounded dull on percussion over the upper third of the sternum, and over a small space a little to the left of it. The respiratory sound was natural. The action of the heart was strong and heaving. Over the precordial region both sounds were heard, the systolic sound being rather distant and coarse. Between the cartilages of the second and third left ribs a single systolic, hollow, rasp-sound was heard; the intensity of this sound diminished in all directions as the ear receded from this point. On regarding it attentively, a slightly elevated spot, rather larger than a shilling, was perceived, which communicated a strong single pulsation and a purring thrill to the hand placed on it. One night he expectorated some blood; and a week after his admission, being agitated and excited in conversation, he suddenly fell back, became deadly pale and insensible; and gasping with slow respiration, whilst his pupils were fixed and dilated, for twenty minutes, he expired.

Inspection. On cutting through the cartilages of the left ribs, a quantity of red-coloured serum escaped. The left pleural cavity was found to contain a gallon of the same kind of fluid; and the left lung was imbedded in an enormous clot. The heart was firm and red, and the parietes of the left ventricle were near the base one inch in thickness. The aortic valves were thickened; and patches of a fibro-cartilaginous consistence and atheroma extended up the aorta.

At the commencement of its arch it was dilated into an aneurism, which might have contained a small fist, in which after three or four lines the coats of the vessel seemed destroyed, or at least were not to be distinguished from the indurated lung, which formed a nest for it. At the upper left corner was an opening, communicating with the left pleural cavity, in which was a recent clot. Slightly adhering to the internal surface of the sac, at its upper and anterior portion, was a soft, mottled, laminated, pyriform concretion as large as a date. The arteria innominata was partially, and the left subclavian artery entirely, plugged up with firm clots. Permission was not obtained to remove the diseased parts.

The aneurism was in this instance detected before the tumefaction, pulsation, and thrill were observed. The absence of the pulse in both arms led to a close examination of the chest.

The stethoscope was applied over a thin flannel shirt, when to the left of the upper part of the sternum a loud hollow rasp-sound was heard, which at the precordial region was so faint as to be with difficulty heard at all; it could not therefore be generated in the heart. Dullness being perceived on percussion at the same spot, the existence of aneurism, containing some coagulum, which extended into the origin of the vessels of the upper extremities, was rendered almost certain. This latter circumstance also proved that it originated in dilatation. When the tumefaction and pulsation were discovered, it became evident that in one spot the coats had given way.

Case XIX.

Sacculated aneurism springing from the ascending aorta, and bursting into the left pleural cavity.

A jobbing smith, æt. 54, stated that when a young man he had been much addicted to drink; that last Christmas he began to feel a pain in his breast, shooting through to his back.

When visited in consultation with Mr. Berry, he complained of some pain in his breast; of palpitations and cough. His expectoration was said to be generally white, occasionally of a yellowish colour. He could lie flat on his back, but not on either side. He had a natural pulse.

A slight elevation was perceived between the cartilages of the second and third left ribs, which was of the size of a half-crown piece. Over this spot a single pulsation was felt. Here also a dull sound was given out on percussion. The respiratory sound was natural. The sounds of the heart were normal above the left nipple, but over the elevation a faint diastolic bellows-sound was heard. It was louder when the patient sat up than when he lay down. The extent and projection of the elevation gradually increased, and the pulsation over it became more liquid. He did not bear well the application of cold. He rather suddenly expired some months after being first seen.

Inspection. The left pleural cavity was full of serum and recent black coagulum. The cartilages of the second and third left ribs were detached from the sternum by erosion of this bone, which, as well as a portion of the left lung, was found adherent to a tumour of the size of a small melon, which was seen lying under and to the left of the sternum. The ascending portion of the arch of the aorta was slightly dilated and studded thickly with atheroma and calcareous scales. At the upper, left, and anterior portion of it was a circular aperture, as large as a shilling, with well-defined edges, leading into a pouch which formed the tumour above described. The inner membrane of the aorta was insensibly blended into that which lined this pouch, but the middle coat terminated abruptly around the aperture. The cellular coat might be seen doubled back upon itself for nearly half an inch, where the pouch rested as it were on the aorta, and then, leaving the vessel, formed the covering of the sac. Where the sac was thus in contact with the aorta, all the coats seemed separated from each other by thickened cellular tissue and atheroma. The anterior and right side of this sac was lined with laminated, tough, discoloured coagulum. At the spot where the cartilages of the second and third left ribs had been detached from the sternum, the blood had passed in front of the ribs, and another small pouch was formed, and was filled with some old and some recent coagulum. This formed the elevation which was observed during lifetime. At the upper part of the back and left side of the large sac, the lung was denuded over a space as large as a shilling, and for some distance around this spot the lung seemed infiltrated with blood, and resembled pulmonary apoplexy in colour, but was much softer in consistence. From this place a channel could be traced for six inches between the pleura and lung, to an opening in the pleura, about six inches from the apex of the lung, and about midway across its posterior portion. Through this opening the blood having previously formed the channel to it, by dissecting the pulmonary pleura from its subjacent lung, had burst forth into the pleural cavity. The appearance of the heart was natural.

This case was obligingly brought under my notice by Mr. Berry. We were unable to obtain permission to remove the aneurism from the body.

The existence of an aneurism was here clearly pointed out by the pulsating tumour, which sounded dull on percussion, over which a murmur was heard which was not audible in the precordial region. The murmur being diastolic led to the belief that the aneurism was sacculated; because it is difficult to conceive in what way a diastolic murmur can be produced in an aneurism by dilatation. During the systole of the ventricles, the edges of the aperture of the sac must have yielded to the force of the blood, and allowed it a free passage into the sac. On yielding in a similar manner to the current passing out of the pouch during diastole, the edges of the aperture would project into the aorta, and thus diminish its calibre, and offer an obstacle to the blood flowing onward under the influence of the contractile power of that vessel.

Case XX.

Aneurism of the arch of the aorta.

A pensioner, æt. 46, formerly 20 years in the horse artillery, and latterly a nurseryman, had invariably enjoyed good health until within two years, when he was one day suddenly seized with a kind of snatching or shooting pain at the back of his head, which was increased by the recumbent posture. It persisted for eight or nine weeks, and was replaced by a sensation of lightness in the head, and once or twice when coming home from work he partially lost his senses and fell down, and he has experienced this sensation occasionally ever since. Four months since he had been engaged for some days in hard trenching among tough roots, when he one day received a strong shock from striking his spade against a stone. He immediately felt a severe pain under the sternum, and pulsation a little to the right of its upper part, so strong that a person whom he requested to feel it thought the heart was beating in that situation. He continued to feel the pain and pulsation for some weeks, when being unable to continue his work, he came to Birmingham by recommendation of his medical attendant, and was admitted into the General Hospital.

When examined, he complained of some severe pain and beating to the right of the sternum when quiet, which were much increased by exertion. He stated that he had lost flesh of late, and that the night before he felt a pricking on the right side of the front of the chest, extending down the right arm. A pulsation was felt over the right clavicle, and a trace of it over the left; none over the top of the sternum. The pulse was natural, but fuller and larger in the right than in the left wrist. Close to the right of the sternum, on a level with the second rib, a slight elevation was seen, the base of which was of the size of a crown-piece, and over this spot was felt a strong single systolic pulsation. The impulse communicated by the stroke of the heart was not great above the left nipple. Dullness was perceived on percussion over the elevated spot. The respiratory sound was natural. The sounds of the heart above and to the left of the nipple were rather confused, and the diastolic sound was accompanied by a long, soft, rasp-sound. Over the lower third and middle of the sternum this sound became a saw-rasp-sound, coarse, hollow, and louder than over the nipple. Over the elevation it was more hollow, but less intense, still diastolic, but the systolic sound of the heart was also heard in all these places. Five weeks after this he complained of much pain and constriction of the chest, and down the right arm. The elevation was, however, less marked and the pulsation in it weaker. Six leeches were applied near the right axilla. In another fortnight he was bled to ℥vj, and a bladder of cold water was ordered to the elevated spot. In a few days he complained of much pain in the chest, and was bled to ℥xvj, with relief. The elevation was much diminished in extent, and the ribs were easily traceable through it. The pulsation was much less liquid, the elevation feeling firmer and more solid. The rasp-sound became much more feeble. A few weeks after this the tumour had nearly disappeared, and his pain and uneasiness were very much diminished. What remained of the tumour was firm and hard, and the pulsations over it had not the least trace of liquidity. He was then discharged.

It is probable that in this, as in Case xiv, the aorta was first dilated, and then ruptured on his receiving a shock. It is doubtful whether the diastolic murmur was generated at the mouth of the aneurismal pouch, or by regurgitation through the orifice of the aorta. The pulsating tumour was enough, however, to indicate the nature of the disease, the blood seeming almost to strike the finger when he was first examined.

Case XXI.

Sacculated aneurism of the arch of the aorta.

A labourer, æt. 53, enjoyed tolerable health until three years since, when he was attacked with severe cough, dyspnœa, and pain in the chest, and in a few days a pulsating swelling of the size of a walnut appeared to the right of the upper part of the sternum. Leeches were applied, and the swelling, pain, and cough disappeared, but considerable dyspnœa remained, particularly on exertion. Eleven months ago he had a similar attack, when he was admitted into the Birmingham General Hospital; and for some time the tumour increased, but eventually receded considerably after he had been leeched, placed under the influence of digitalis, and had cold applications to the tumour. He was discharged. Last week he experienced another attack, and on one or two occasions he spat up a little bright red blood.

On his readmission into the hospital he was seen to be emaciated, was pale and sallow; he had an anxious haggard expression of countenance. He complained of total loss of appetite, great dyspnœa, increased by exertion, severe pain under the sternum, darting up to the right clavicle and to the right hypochondrium; he lay easiest on his right side. Between the first and second right costal cartilages a tumour projected which was seen to pulsate, and on which pulsation by no means liquid was felt with the fingers. It covered a space about the size of a crown-piece. The upper part of the sternum was slightly projected forwards. The tumour sounded dull on percussion. The respiratory sound was rather feeble under both clavicles, and was in places accompanied by cooing sounds. The action of the heart was heaving, and both its sounds rather muffled and prolonged. No sound was heard over the tumour.

The patient was, after a time, relieved by cold applications to the tumour, &c., and has not since been heard of.

Although the pulsation in the tumour was not very liquid, it was sufficiently so to prove that it was not propagated from the aorta by any solid or liquid lying before it. The tumour was clearly a sacculated aneurism containing some fibrinous clots. Its pulsations were propagated over the whole of the upper part of the chest.

Case XXII.

Sacculated aneurism springing from the ascending portion of the arch of the aorta.

A shopkeeper, æt. 38, had, during the year 1837, experienced more or less of pain in the front of his chest. In November of that year he had an attack of illness which was said to present the character of bronchitis. Soon after this he felt pains in the thorax running down the right arm, which he supposed to be rheumatism, his right arm often feeling as if it were asleep. In a short time after this his medical attendant perceived a swelling to the right of the upper third of the sternum, over which he felt distinct fluctuation and a strong pulsation. In November, 1838, he took cold, and the pain in the chest became aggravated, being most urgent when he was sitting quiet.

The tumour had now attained the size of half a large melon. He was placed on a rigidly spare diet, was bled repeatedly, and ordered digitalis. On visiting him in February, 1839, a very large swelling was seen over the upper half of the right side of the thorax, and a very liquid pulsation was felt over the whole of it, with heaving impulse. All the sounds of the chest were natural, but the double sounds of the heart, which were clear, were heard distinctly over the tumour. A bladder of cold lotion was ordered to be kept almost constantly over the tumour; great quietude was enforced, and mild sedatives and nutritious diet were ordered.

In a few weeks the tumour was seen to decrease, and his diet was gradually improved. In August the swelling was reduced to the size of half an orange, and felt firm and hard, the pulsation being liquid only in one small spot. The cold application was discontinued, and animal food and gentle exercise allowed every day. Towards the middle of the month he was seized with a violent attack of acute bronchitis, which carried him off in eight days.

Inspection. A smooth spheroidal tumour, with a dense, shining, aponeurotic covering, was seen lying in the upper part of the right side of the thorax and under the upper third of the sternum; it was as large as a small melon, and had pushed aside and compressed the upper lobe of the right lung. The substance of the lungs was healthy, and the compressed portion was easily inflated. The lining membrane of the bronchial tubes was livid, puffed, and in some places softened; they contained much ropy mucus. The tumour adhered to the sternum and to the cartilages of the second and third right ribs, and partly to the second rib, which had become detached from its cartilage by erosion. The tumour was found to be an aneurismal sac, springing from the right and anterior portion of the ascending part of the arch of the aorta, with which it communicated by an oval orifice. With the exception of a small portion immediately behind the sternum, every part of the sac was lined with dense firm coagulum from half an inch to two inches in thickness. There remained room in the sac for about eight ounces of fluid. The great size of the tumour, and its extension on the right side of the chest, had caused it to push the large vessels further back from the sternum than usual. The left vena innominata and the superior vena cava lay in deep grooves on the tumour, and each contained coagulum. A prominence in the interior of the sac was found to correspond with these grooves. The descending aorta, innominata, carotid, and subclavian arteries were all healthy.

The preparation is in the Museum of King's College, London.

One glance revealed the nature of the affection. It was very interesting to watch the gradual diminution in the size of the tumour, and its increased solidity from the formation of coagulum within it. Before the fatal attack of bronchitis the patient was enabled to attend to his shop.

Case XXIII.

Sacculated aneurism springing from the ascending aorta, and bursting into a tubercular cavity in the right lung.

A married female, æt. 27, two years ago had a severe illness, which was considered a liver complaint, and for which she was salivated, since which time she had never felt well, and had a constant singing in her ears, with deafness and a severe cough. At the time of that illness she had great pain under the xiphoid cartilage, which shot up the right arm and shoulder; and also a beating at the heart, which seemed to extend up her throat, and at times interrupted her swallowing. For twelve months she had perceived a swelling to the right of the upper third of the sternum, which had greatly increased in size during the last two months.

Being admitted into the Birmingham General Hospital, dyspnœa on movement was most urgent, and threatened suffocation; she was very deaf, and complained of noises in the head, and of great pain about the precordial region, striking up to the right shoulder and down the right arm; of a troublesome cough, accompanied by white frothy expectoration, and of inability to lie on the left side, without dyspnœa and great increase of the cough. Her pulse was small and feeble, but regular and very frequent. Both a pulsation and a purring thrill were felt above each clavicle and above the sternum. To the right of the upper third of the sternum a swelling of the shape and size of half an orange, resting on an outer slightly elevated ridge about two inches wide, was perceived. Over the middle of this tumour a single and very liquid pulsation was felt, and a purring thrill, seeming as if the fluid within the tumour struck the finger at each pulsation through a very thin wall. Nearly the whole of the upper half of the right side of the chest sounded duller on percussion than that of the left side, more especially that portion of it which was occupied by the tumour. The respiratory sound was nearly absent over the upper third of the front of the right side of the chest, and rather blowing and yet more feeble at the upper third of the back part of the right side than over the corresponding portion of the left side. No rattles were detected. The action of the heart was natural, and also its sounds. A well-marked, but not intense, rough, double saw-sound was heard over the tumour, but was much more distinct during the systole of the heart than during its diastole.

She was ordered to be kept perfectly quiet, to have a light, nutritious, but unstimulating diet, including meat daily, to take some camphor and hyoscyamus, and to have a bladder of refrigerating lotion kept over the tumour as constantly as she could bear it. Under this treatment, with two or three applications of four leeches to the epigastrium and to the right axilla, and with the occasional employment of belladonna frictions over the chest, the dyspnœa and anxiety in a great measure disappeared; the pulse became less frequent, the tumour sensibly decreased, and in four months it was reduced to the size of half a walnut, inclusive of its edges, and in another six weeks the chest was nearly level. What could be then felt of the tumour was firm and hard; the strength of the pulsation was much diminished; it no longer seemed liquid, but felt distant and less sharp; the purring thrill was gone. The saw-sound was become very feeble, and only diastolic. One day she vomited, as she said, a mixture of matter and dirty-coloured lumpy blood, amounting to about an ounce.

She remained much in the same state for nine months, suffering occasionally from severe headaches and paroxysms of great pain in the epigastric region, accompanied by pyrosis and loss of appetite, and acceleration of the pulse, a state which was generally relieved by ten-minim doses of the diluted hydrocyanic acid, and some leeches. The tumour occasionally increased in size after her having taken cold and coughed violently. Having been for some time at home, she was again received into the hospital after an attack of influenza, when the tumour was found to have slightly increased in size, and she could not lie down flat without great dyspnœa. Her cough was troublesome, and she expectorated much frothy mucus. She had intense pain, extending from the sternum up the right side of the neck. The pulsation in the tumour was strong, but not liquid; only a faint sound was heard over the tumour, alternating with the heart's impulse. The back part of the upper half of the right side of the chest was duller than before, and the respiratory sound was much more feeble over the whole of the right side than over the left, and there was much mucous rattle over both sides of the chest. This bronchitis was soon relieved, and then she varied little until she left Birmingham for Ambleside, where she was attended by Mr. Fell, and was seen occasionally by Dr. John Davy, and where she died suddenly, suffocated with blood, about two years after her first admission into the Birmingham Hospital.

Inspection, by Dr. John Davy and Mr. Fell.

Both lungs adhered to the chest. The left lung was small, but in a healthy state; there was a little blood here and there in its bronchial tubes. The left lung was dense, and there was much dark coagulated blood in its tubes. There were large patches of gray induration in its middle lobe. In the upper lobe was much of the same matter, containing tubercles in it, and also several tubercular excavations, one of which was of the size of a chesnut, and was quite at the apex of the lung. A large tumour, about the size of a child's head, lay under the upper part of the sternum, extending a little to the left of it, but principally to the right, where it adhered to the diseased lung, and to the sternum and costal cartilages. This was found to be an aneurismal pouch, springing from the right side of the ascending aorta just above the valves. It ran up the outer edge of the arteria innominata, which was not involved in it, so that the origin of this vessel had a valvular appearance. This sac was nearly filled with fibrin, the outer layers of which were very firm and discoloured. In one spot there was an ulcerated opening which would have admitted a crow-quill, and which communicated with a small tubercular cavity. A portion of the sternum in contact with the sac was eroded; and on the right of it, over a space of the size of a crown-piece, the sac was bounded by the common integuments, and produced a slight external protuberance. The heart and its valves were healthy.

The preparation was kindly sent to me by Mr. Fell, and is in the Museum of King's College, London.

The nature of the affection could not here be mistaken. A pulsating tumour, a purring thrill, and a double murmur distinct from the sounds of the heart, at the upper part of the right side of the chest, rendered the diagnosis certain. The aneurismal sac being in part bounded by the integuments, the finger was enabled to trace what was going on within it. In proportion as its size diminished, so did the liquidity of the pulsation, and the increase of solid matter between the finger and the blood could distinctly be perceived.

The post-mortem appearances seem satisfactorily to explain why the murmur was at one time only diastolic. On the cessation of the systole of the heart, the sac would compress the ascending aorta and arteria innominata, and thus offer an obstacle to the blood flowing onwards, which would not exist when these vessels were fully distended by the heart's action; and also the edges of the orifice of the pouch giving way to the force of the blood, would favour its entrance into the pouch, but would obstruct its onward flow through the aorta during diastole, by protruding into the vessel, as in Case XIX.

Case XXIV.

Sacculated aneurism springing from the summit of the arch of the aorta—Rupture of external integuments.

A japanner, æt. 39, applied for advice, and stated that he had experienced more or less pain in his head for the last six months, from exposure to cold; and that at the time of application it was very severe and constant. The pulse was moderate. The edges of the tongue were red, and its middle was covered with a thick, yellowish-brown coat. The appetite was impaired; he could not sleep. His bowels were very much confined. The respiratory sounds and those of the heart were natural. In ten days he found himself greatly relieved.

Four months after this he again applied for advice, stating that during the whole of the previous month he had felt sharp cutting pains between the shoulders, and that towards its close he perceived a swelling above the sternum, inclining towards the left side, in which he occasionally felt severe pain. It was about the size of a hen's egg, and irregular in shape, owing to the cicatrix of an old abscess in the middle of it, at which point the skin was of a purple colour. The tumour rose up from behind the top of the sternum, and bent forwards over it. Fluctuation was very manifest in it, and strong and liquid pulsations were felt all over it. The pulse was natural, and the pulsation of the carotids very moderate. The action of the heart was also rather feeble than otherwise. The chest sounded clear on percussion, the precordial dullness being more limited than usual. The respiratory sounds and those of the heart were quite natural, nor was there any unusual resonance of voice, or any sound heard over the tumour. He was much relieved by sedatives, and by the application of cold lotion to the tumour. Still it rapidly increased, until it was larger than the fist.

In six weeks' time blood began to ooze from the purple spot upon it, which ceased on the application of mucilage and creosote, which was applied by Mr. Bindley, who saw him at this time. It continued to ooze occasionally, and he became much emaciated, was sometimes troubled with dyspnœa, and was very feeble.

A month later the tumour seemed harder, fluctuation was less marked in it, and the pulsation over it was less liquid, but strong. Soon after this a gush of blood burst forth, and he died.

Inspection. The tumour was found firmly adherent to the top of the sternum, which was removed with it. The heart was very small but healthy, as also were the lungs. The inner surface of the aorta was smooth; it was uniformly dilated. At the anterior and upper part of the arch was an oval orifice, the edges of which were smooth and rounded; it was as wide as a half-crown piece, in the direction of its long axis, which was from side to side, and opened into an aneurismal sac, nearly spheroidal, the external circumference of which measured twelve inches. A smooth membrane, that seemed continuous with the inner membrane of the aorta, entered the sac for about half an inch all around the orifice, and beyond this layers of red coagulum adhered to the walls. This kind of clot completely filled up the pouch, but a circuitous channel from the aorta to the external ulcerated opening could readily be traced.

The preparation is in the possession of Mr. Bindley.

The study of such aneurisms as this, springing from the upper part of the arch of the aorta, and rising above the sternum or clavicles, is extremely interesting in a surgical point of view, because they have sometimes been mistaken for aneurisms of the subclavian and carotid arteries; a mistake of the highest moment, since it might be the means of inducing a surgeon to commence an operation which, to say the least, would soon be found impracticable, and might hasten the end of the patient.

A case of this kind, recorded by Burns in his work on the Surgical Anatomy of the Head and Neck, was mistaken for aneurism of the subclavian artery, "by several of the most distinguished practitioners in Edinburgh, and almost every surgeon in Glasgow," some of whom strongly advocated an operation. After death, the aneurism was found to arise from the aorta, and included a considerable part of the arteria innominata. It mounted considerably above the sternum, pressing in its ascent the descending vena cava to the right, and the trachea to the left side.

A somewhat similar case is recorded by Sir Astley Cooper, who mistook the disease for aneurism of the carotid artery. The tumour had the shape of a Florence flask inverted, and arose by a very narrow neck from the arch of the aorta, between the roots of the left subclavian and the left carotid arteries. It rose up between these vessels, and appeared at the root of the neck, so that it resembled an aneurism of the carotid artery more than one of the aorta.

The aorta in Case xxiv not having been submitted to microscopic examination, the existence of atheroma was not proved. The sac might have originated either in a partial dilatation, or rupture of the coats, which were traceable for about the distance of half an inch into it. The aneurism once formed, would rapidly gain the surface above the neck, from the circumstance of the deep-seated fascia which extends from the sternum and clavicles to the lower edge of the thyroid gland having been partially destroyed by the abscess which had been formed in boyhood, and the cicatrix of which was seen in the centre of the tumour; and this accords with the history of the case. Before, however, it gained the surface, it must have interfered with the nerves distributed to the shoulders and arms, and thus have produced the pains described, which pressure would be diminished in proportion as a free passage was opened in an upward direction. So also, while yet within the cavity of the thorax, the veins of the head must have been more or less compressed, and thus the violent headaches of which the patient first complained may have been produced. The tumour was at once recognised as a sacculated aneurism of the aorta, being traceable by pulsation a considerable distance behind the sternum with the finger, and being evidently filled with fluid blood. It was some time before cold applications were successful in causing coagulation of blood within the sac. Before death, however, this had taken place to a considerable extent, and the life of the patient might have been protracted for some time, had it not been partly for the attenuation of the integuments by the abscess in his younger days, and partly from the soft nature of the clot, which prevented its adhesion to the sac.

None but the ordinary sounds of the heart were heard over or about the tumour. The heart was small, and its action feeble, and therefore not such as would easily generate an abnormal murmur. Nor was the uniform dilatation of the aorta, which was smooth, likely to give rise to any murmur, although it might have impressed a particular character on one originating in diseased aortic valves. These, however, were healthy.

CASE XXV.

Mixed aneurism of the arch of the aorta compressing the superior vena cava—Bronchitis.

A married female, æt. 55, had long laboured under troublesome cough, with copious yellowish and gray-coloured expectoration, and wheezing respiration, with some dyspnœa. In this state she applied for advice, when the chest sounded clear on percussion, but the respiratory sound was coarse, and was accompanied by cooing and bass-viol sounds, and in some places by uneven, moist rattles. The sounds of the heart were clear, but distant.

She was lost sight of for three months, when she was visited in bed. She stated that she had not been well since she was last seen; that dyspnœa had gradually increased; and that a "beating substance" had risen above the sternum, which had receded during the last week. Her medical attendants, Mr. Green and Mr. Carter, both stated, that during their attendance a softish rasp-sound was heard at the top of the sternum. She was labouring under most distressing dyspnœa, and was not able to assume the recumbent position for a moment. Her breathing was accompanied by a loud whistling, as if the trachea were compressed, and by a large, moist, tracheal rattle. The top of the sternum, for a distance of two inches down, was pushed out and bent forwards. Above this a fulness could be perceived, but no pulsation could be felt. There was expectoration of thick yellow mucus. There was œdema of the head, neck, and arms, but of no other portion of the body. The sounds of the heart, particularly the systolic sound, were feeble but distinct, and were heard rather louder over the top of the sternum, and over the upper third of the front of the left side of the chest, than in the precordial region, when the breath was held. The upper third both of the sternum and of the front of the left side of the chest sounded very dull on percussion. The respiratory sound was lost in the tracheal rattle. Six hours after, on attempting to lie down, she suddenly expired.

Inspection. It was found, on removing the sternum, that at its upper part, for two inches in length, it was closely adherent to a subjacent tumour. This portion was therefore separated from the remainder of the bone, and left adhering to the tumour. This was found extending a little to the right of the sternum, but principally backwards, where it lay on and compressed the trachea, and to the left, where it greatly encroached on the upper third of the left lung, and was double the size of the fist. This portion of the left lung was carnified, and of a dark slate colour. On being cut into, numerous drops of thick primrose-coloured fluid exuded from the bronchial tubes. It adhered most closely to the ribs. The lining membrane of all the bronchial tubes was pale and thickened. The heart was of a natural size, rather pale and flaccid. The valves on its left side were slightly thickened, but acted well. The left vena innominata and the superior vena cava were found closely adherent to, and compressed by the tumour, the latter being lodged in a superficial groove on its surface. The tumour was found to be a dilatation of the aorta, which commenced a little above its origin, and was continued to the origin of the left subclavian artery. The coats of the aorta much thickened and corrugated, with deposits of atheroma and fissures, could be traced for some distance, but were deficient at the upper and anterior portion. Here there was an adhesion to the upper portion of the sternum, which was denuded of periosteum. To the right of this and above it, traces of the inner coats of the vessel were also lost, and over the whole of this space there was a large mass of firm coagulum. This coagulum filled half the sac, the remainder of which might have contained six ounces of fluid. A clot of fibrin hung like a flap over the origin of the innominata, and when pressed against it closed it.

The preparation is in the Museum of King's College, London.

There was here clearly a tumour in the mediastinum, compressing both the trachea and the superior vena cava. If the accuracy of the statement of the patient could be relied on, this tumour was an aneurism, very similar to that in Case XXIV, which had decreased in size, owing to the deposition of fibrinous clot within it. So again, if sound had at one time been engendered in the pouch, it must have ceased from the decrease of the muscular power of the heart, as she approached her end, for the deposition of coagulum in it would have tended rather to increase than to diminish the murmur. Had the patient been examined at an earlier period, it is probable that a pulsating tumour would have been felt behind the top of the sternum, and a rasp-sound would have been heard in the same situation. As it was, however, the nature of the affection was apparent.

Of the twenty-three cases here recorded, thirteen were seen before the aneurism had reached the surface of the chest. In seven of these the nature of the affection was recognised; in three the existence of a tumour, which compressed the trachea, was ascertained, but its precise nature could not be determined. Of these three it may be remarked, that in Case VII, no opportunity of careful examination was afforded; that in Case VI, had the expectorated blood been preserved, in all probability the nature of the tumour would have been revealed, as in Case IX; and that in Case VIII, the diminution of dullness and pulsation, after a copious hemoptysis, could only be explained on the supposition that the tumour was aneurismal. There remain then only three in which it can be positively stated that there were no signs whatever characteristic of the disease; and these were cases in which the sac burst into the pericardium before they attained a size larger than that of a walnut,—Cases III, IV, and V.

I might here give the particulars of thirteen cases of aneurism occurring in the practice of others, in eleven of which notes were taken by myself, both of the signs observed during lifetime, and of the appearances after death, and in two who died suddenly of the latter only; but, as they more or less resemble some one of the cases here detailed, it would be useless to record them at length. I shall, however, make use of them in testing the value of the diagnostic signs of this disease, in conjunction with those above related.

I might also bring forward several cases in which I have strongly suspected that aneurism has been present; but in which, either from the circumstance of the patients being still alive, or from their having been lost sight of, I have had no opportunity of actually proving the correctness of my opinion.

The following is one of such cases, but I shall relate no more, as they cannot be used for the purpose of illustrating the diagnosis of aneurism.

Case XXVI.

Aneurism of the arch of the aorta.

A shoemaker, æt. 27, was sent to me for examination by the surgeon of a club, who hesitated to pass him for admission. He professed to be in excellent health, and asserted that he had always been so. Slight dullness on percussion was perceived a little to the right of the upper third of the sternum. The respiratory sound was quite natural, as also were the sounds of the heart. Over the spot where the dullness was perceived, was heard a well-marked, systolic, hollow rasp-sound, which did not extend far. On pressing down the finger between the second and third right ribs and their cartilages, a faint pulsation and a purring thrill could both be distinctly perceived. No pulsation could be detected over the clavicles or sternum.

His life was of course pronounced unfit for insurance, and I saw no more of him.

The single systolic sound heard in this case might, as in Cases xxix and xxx, have been produced by constriction of the aorta, but the dullness on percussion proved the existence of some solid substance. If this were solidified lung, it must have been confined to one small spot; and in that case it was not likely that it would have propagated a purring thrill to the fingers, although it might have propagated a faint pulsation. These circumstances, taken in conjunction with the absence of all signs of disease of the lungs, rendered the existence of aneurism almost certain.

CHAPTER IX.

DIAGNOSIS OF THORACIC ANEURISM.

In proceeding to test the value of the rules that have been laid down for our guidance in the discovery of aneurism of the thoracic aorta, it will be desirable in the first instance to consider each sign separately.

Pulse. It may easily be imagined that when the whole or part of the blood which issues from the left ventricle has to pass through an aneurismal pouch, its motion may be modified in such a manner that a shock of a peculiar nature may be communicated to the finger laid on an artery.

Some writers have described the pulse as having a resilient character in certain cases of aneurism, which they attribute to a second impulse given to the blood by the reaction of the aneurismal pouch after its distension. Undoubtedly such a pulse is sometimes felt, and the cause assigned for it is probably the true one. But it is no less certain that a very similar kind of pulse is often found in certain diseases of the heart uncomplicated with aneurism. Now supposing that we were enabled by a very nice sense of touch, and very long practice, to distinguish between these two kinds of pulse, it is clear that such a sign would be almost valueless from the difficulty of appreciating it, and the rarity of the aneurismal variety. But it is one thing to distinguish between the extremes of the pulse in respect to strength, rapidity of stroke, fulness, and frequency; and another to detect the variations of the pulse which result as well from aneurisms of all sizes and shapes, as from certain affections of the walls and valves of the left side of the heart; and the more expert the practitioner becomes in detecting the difference of the one from the other, the more will his observation have convinced him that his skill in this respect is of little practical value, either as regards diagnosis or treatment. We shall not be surprised at this when we consider the different forms under which aneurisms present themselves to our view; in some cases having thin and elastic, in others firm, inelastic walls, or adhering in such a manner to the neighbouring parts as to be incapable of expansion; associated sometimes with a powerless, softened heart, at others with this organ largely hypertrophied and vigorous; some of them nearly filled with solid layers of coagulum, and others containing liquid blood alone. These opposite states must each affect the pulse in a manner different the one from the other, and in many cases produce on it effects similar to those which result from certain forms of diseased heart, which it is unnecessary here to describe.

A valuable sign, however, is sometimes furnished by the absence of the pulse at the wrist, and in the axilla; because this frequently results from a plugging up of the origin of the vessel by a clot connected with an aneurismal pouch. But it may arise from a deposition of atheroma, or the formation of horny patches. In Case XXXVIII the right pulse was very small, and a rasp-sound and purring thrill were discovered under the right clavicle, which signs were produced by a narrowing of the origin of the arteria innominata by atheroma. In Case XVIII, the patient having been admitted for apoplexy, the nature of the thoracic affection had been entirely overlooked, when, finding the absence of a pulse in the right wrist noted by the clinical clerk, I was induced to explore the chest, and immediately discovered the aneurism. This sign in Case XIII not only led to an exploration of the chest, but materially assisted in the formation of the diagnosis.

There is another state of the pulse which may sometimes assist us in preventing aneurism, or detecting it at an early period of its development. This is the sharpness and rapidity of stroke which results from a diminution of the elasticity of the coats of the arteries. This once detected, the medical attendant would not only carefully search for aneurism, but would also place his patient on such a plan as would be most calculated to prevent arterial dilatation or rupture, which might so easily occur under these circumstances.

Unless therefore under very peculiar circumstances, as in the case above mentioned, the pulse cannot be considered as affording very valuable assistance, either in the discovery or treatment of thoracic aneurism.

Pulsation. When a pulsation is both seen and felt over a circumscribed prominence on the chest, it has always, in my experience, denoted the existence of an aneurism. It is, however, possible that an abscess lying over the aorta might give rise to similar signs. But when pulsation is found without any external prominence, it may arise from various other causes. "Underneath the sternum or ribs at the superior part of the chest," Dr. Hope[1] is of opinion "that it is one of the least equivocal signs of aneurism, although it is not without ambiguity." He states that in this situation (below the clavicle) pulsation is produced by sacculated aneurism, and that when it is felt above the sternum and clavicles it generally results from dilated aneurism. Experience has taught me to attach but little value to such pulsations as diagnostic signs of aneurism, except when they are either limited to a circumscribed spot near the upper half of the sternum, or are at their maximum there. For besides being occasionally caused by tumours and enlarged glands lying on the aorta, very strong pulsations are frequently seen to arise from hypertrophy of the heart alone; whilst, on the other hand, aneurisms of considerable size sometimes approach close to the surface of the chest, and even adhere to the sternum without producing any pulsation in that spot. In Cases XXXVII and XXXVIII, and numerous others of great hypertrophy of the left ventricles, such pulsations were seen; whilst in Cases VII, X, and XI none existed; although in Cases X and XI an aneurismal tumour as nearly as possible touched the sternum, and in Case VII actually adhered to it. No pulsations were felt above the clavicles in Cases VI, VIII, and IX, in all of which an aneurism adhered to the trachea, which might have been expected to propagate its pulsations upwards; nor were there any immediately above or below the clavicles in Cases XIII, XVIII, and XIX, although a pulsation was felt on the spot where the aneurism reached the surface of the chest. Out of thirty-two cases of thoracic aneurism originating without the pericardium, there were only ten in which pulsation occurred immediately above or below the clavicles; and it is remarkable that pulsations in this situation were observed in but one of those cases in which the aneurismal tumour did not reach the surface of the chest at the time of examination. It is probable therefore that some writers have described what they have supposed ought to exist rather than what they have actually observed. For when the aorta is dilated it is generally at the summit of the arch, against which the current of the blood impinges strongly, and hence it may have been supposed that there would be a tendency to the production of pulsations above the clavicles. A sacculated aneurism, on the other hand, generally stretches towards the surface of the chest below the clavicles, except in such Cases as XXIV and XXV, and consequently pulsations are to be looked for in that situation. Such may be the case when pulsations exist; but observation has taught me that the formation of pulsations, when the aneurism does not touch the surface of the chest, in a great measure depends on the force of the heart's action. In one of two cases wherein there was an equal amount of uniform dilatation of the aorta, pulsation was distinctly felt on pressing the finger deeply down behind the sternum; and in the other no pulsation could be detected. In the former case the heart was large, firm, and red; and in the latter it was in a state of brown fatty degeneracy. Thus in Case XXV, when the vital powers were nearly extinct, no pulsation could be felt over the aneurismal tumour; and in Case VIII the force of the pulsation was much diminished after copious hemoptysis. This accounts for the frequency of pulsation above the clavicle in cases of hypertrophy of the heart without aneurismal tumours.

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[1] Hope, p. 439. 3d edit.

When pulsations of the arch of the aorta are sought for by the finger pressed down behind the upper part of the sternum, great care must be taken to avoid touching either of the carotid arteries, as an error would thereby be induced.

Pulsations may also be perceived in other parts of the chest, which are not produced by aneurisms. Thus a feeble pulsation is sometimes seen and felt between the second and third left ribs, which arises from the impulse of the heart communicated through the appendix of the left auricle, greatly dilated, and in contact with the chest at that spot, as in Cases LI, LVIII, &c. So also pulsations are occasionally seen considerably to the left of the precordial region, which arise from adhesion between the pericardium and the ribs, either directly, or through the intervention of the lung, as in Case XXXV. It is hardly necessary to remark that the seat of the pulsation, produced by the action of the heart, varies with the position of that organ; which may be altered by pleuritic effusion or other causes.

When pulsation takes place over an elevated spot, its nature is worthy of every attention, varying as it does from an obscure dull heaving to a sharp quick stroke, which gives the idea of liquid almost touching the finger. In the former case, more or less coagulum lines the sac, and is interposed between the finger and the blood; and in the latter case the walls of the sac at this point are thin and elastic, and the blood impinges directly upon them. In this manner the formation of a coagulum within the sac may in certain cases be traced by the touch—as in Cases XX, XXII, XXIII, and XXIV. In Case XIV, whilst the tumour was approaching the surface, and some lung intervened between it and the walls of the chest, the pulsation was heavy and dull; but became very liquid on the blood reaching the integuments. The importance of duly studying the nature of the pulsation will be fully recognised, when we consider the treatment of aneurism.

Thrill. Purring thrill denotes the proximity to the walls of the chest of a cavity through which blood is passing, that has been thrown into agitation by some unusual obstacle to its course. Hence it is perceived when the heart is dilated, and there is at the same time obstructive disease of its valves; also, when the coats of the aorta are dilated and diseased; and, although in a less degree, when a sacculated aneurism, which communicates freely with the aorta, approaches the surface of the chest. In the latter case the larger the sac the less marked will be the thrill, from the increased force required to put the whole of its contents into motion.

It is clear that the strength of the thrill, like that of pulsation, depends in a great measure on the force of the heart's action. Dr. Hope found it much less marked in sacculated than in dilated aneurisms. In Case XIV it existed before any elevation was perceived, and proved to be a diagnostic sign of great value. So also in Case XV it was perceived without any elevation, and in connexion with a very loud rasp-sound; it was so remarkably strong, and appeared to be produced so immediately under the finger, that its seat was almost necessarily referred to the pulmonary artery; and by these two signs alone the nature of the affection was discovered. In Case XVIII it existed over the elevation; in Case XXIII, over the tumour and over each clavicle; and in Case XII, over the external prominence, and behind the sternum. It also existed over a circumscribed space to the left of the sternum in Case XXIX, which will presently be detailed, and being coupled with a murmur, led to a suspicion of aneurism, which was not confirmed; having been in fact produced by constriction of the aorta; although that vessel was very slightly dilated behind the contraction, and was in a position much higher than natural.

The purring thrill is frequently propagated along the course of the carotids, when it is produced either in the mouth of these vessels, as in Case XXXVIII, or at the sigmoid valves, as in Case XXXVII, by a strong current of blood. None would of course construe this into a sign of aneurism.

General signs of thoracic aneurism may also be derived from its effects on the contiguous parts of the body. These are the heart, arteries, veins, and nerves of the chest, the trachea, bronchial tubes, and lungs, the œsophagus and vertebral column.

Heart. Palpitations, syncope, dyspnœa, irregular action and pain, may be induced by the pressure of an aneurismal tumour on the heart, or by the obstacle which a considerable dilatation of the aorta would oppose to the flow of the blood from the heart. But all these symptoms may result from disease of the organ itself, and therefore they are of no value as diagnostic signs of aneurism.

Vessels. The arteries of the neck and arms are sometimes compressed in a peculiar manner. Thus in Case XXIII the sac crept up along the arteria innominata, and when distended must have compressed the vessel. Hence there would be inequality in the circulation, which possibly gave rise to the headaches with which the patient was tormented. So, also, in Case VIII, the left carotid artery was compressed from behind; and this patient also suffered from intense headache, particularly when lying on his right side. As headaches are very common, and arise from a variety of causes, they would not serve to point out the nature of the affection under consideration. Pressure on the vessels of the lungs is interesting in a pathological point of view, but as it would give rise to a state of the lung which might arise from other causes, it furnishes no diagnostic signs.

When the descending vena cava, hewever, is compressed, a curious and valuable sign is developed; its existence being revealed by anasarca, confined to the head, neck, arms, and chest. For pressure on the descending vena cava, which is the only trunk that unites the veins from these parts, could alone give rise to such a state. The existence of a tumour, therefore, in the immediate vicinity of this vessel, which is not long, is thus clearly indicated. Such tumour, however, is not necessarily an aneurism, as is proved by the following case.

Case XXVII.

Carcinomatous tumour compressing the descending vena cava, and producing œdema of the head and arms—Similar tumour around the stomach—Cyst in the brain.

A gilder, æt. 43, having felt poorly for some months, which he attributed to over-exertion in his business, consulted Mr. Wickenden, complaining of severe pain in his head, and uncomfortable sensations at his stomach. His breath was fetid, his gums spongy, and his bowels constipated. His pulse was slow, but natural in other respects. The sounds of the heart were heard over the whole of the front part of the chest. He thought he should be well if he could live without eating, since food caused him great uneasiness. Soon after this a tremulous motion of the hands was observed, and he complained of great pain in his head, shoulders, and arms. Having joined Mr. Wickenden in consultation, I continued to attend with him until the death of the patient. His chief complaint was of intense pain in the head.

The pupils were small and contracted, and the iris inactive. The pulse 40. The pain in the head was slightly relieved, and the pulse rose to 70 under the use of the cold douche. He went to the seaside for three weeks, during which time it appeared that he had three epileptic fits. On his return his symptoms seemed aggravated. His memory of words was affected, and his sight became slightly impaired. He was very irritable, and wandered in his mind. In a few days the pain between his shoulders became excessive, and he was rather violent. Strong narcotics relieved this pain, but that in his head remained, although blisters were repeatedly applied down the spine.

Six months after his first seizure he had no pain, except in the head; but dyspnœa came on, and œdema of the lower right eyelid. There was slight dullness to the right of the upper part of the sternum. The respiratory sound was natural; the sounds of the heart were extended but normal. The pupils were now dilated, the pulse small and quick, 120. Œdema of the head, neck, arms, and upper part of the thorax came on. The sounds of the heart were heard louder, the respiratory sound more feeble, and the sound given out on percussion a little duller than elsewhere, at the right of the top of the sternum. There was whistling respiration as if the trachea were compressed. He could not lie down, but spent his time leaning forwards. He complained of pain in the head, and wandered at times. Slight œdema of the legs and scrotum next appeared, and when seen again, he was in a profuse perspiration, leaning forwards, and with the greatest difficulty drawing in his breath; and he complained of a pain in the head.

He died in this state four months after he was first seen, and about seven months after his first seizure.

Inspection. The body was opened by Mr. Wickenden and Dr. Mackay during my absence from home, and the following appearances were noted by them. The face, neck, arms, and breast were very œdematous, the legs and scrotum slightly so.

In the inferior and posterior portion of the left hemisphere of the brain was a cyst of the size of a hen's egg, which rested on the tentorium cerebelli. It was thick, and was surrounded by small tumours, varying in size from that of a pea downwards, hard, white, and lardaceous. It contained about an ounce of clear fluid, and around it the brain was softened.

The right pleural cavity contained three, the left two pints, and the pericardium two ounces of clear fluid. The heart was dilated, large, soft, and flabby. A white, hard, lardaceous tumour, with a spot or two of softening in it, stretched from the bronchial glands to the right edge of the upper part of the sternum and sternal end of the right clavicle. The vena cava superior was imbedded in it and flattened. The lesser curvature, and indeed the greatest part of the stomach, was embraced by a solid mass of a similar kind to that found in the chest, which also invaded the mesenteric glands, and the liver contained a mass, rather larger than a walnut, of the same deposit.

The signs of a mediastinal tumour were unequivocal, but it was not supposed to be an aneurism, because although it evidently was in contact with the surface of the chest to the right of the sternum, and conveyed the sounds of the heart with unusual clearness to the ear, yet there was no trace of pulsation over that spot. The same sign was observed in Cases XI and XXV, but there were other signs in these cases which proved the tumour to be aneurismal.

The absence, however, of anasarca of the upper part of the body does not prove the non-existence of a tumour near the descending vena cava, because there was a tumour in this situation in Cases X, XIV, XXII and XXIII, but the vessel was not compressed by it.

It is said that the thoracic duct is sometimes thus compressed, and that inanition follows from the flow of the chyle into the blood being interrupted. Possibly such was the case to a certain extent in Case XIII, but this point was overlooked in the inspection of the body.

Nerves. The pains so often felt in the chest and down the arms in thoracic aneurism are doubtless caused by the nerves being in some degree implicated. But as such pains are not confined to aneurism, they are not characteristic. Still they are not without value, as they induce patients to apply for advice, and thus pave the way for the discovery of this affection, if it exists, by means of other more certain signs.

The recurrent nerve is sometimes involved, and the voice then becomes affected in consequence of paralysis of the muscles of one side of the larynx taking place.[1] Dr. Todd has related a case of this kind. It also occurred in Case VIII. The position of the tumour in Case IX was such that it probably occurred there also, especially as the voice was reduced to a whisper some time before death, but this point was overlooked in examining the body.

Trachea and lungs. The chief symptom produced by pressure on the trachea and lungs is dyspnœa, which, as it may arise from so many other causes, is of no value as a diagnostic sign of any one disease. It may, however, be so modified by peculiar circumstances as to become valuable. For instance, if it should only occur, or be greatly increased when a person is in the upright or recumbent position, as in Cases X and XXVII, it would indicate that some solid body fell back on the trachea and lungs.

This effect might, to a certain extent, be produced by a dilated heart, as in a case related by Morgagni.[2] A careful exploration of the precordial region, however, would lead to the discovery of such a cause as this. When the trachea is thus compressed, it is sometimes pushed backwards from the sternum, as in Case VI, or to one side of the mesian line, as in Case VIII. A loud whistling sound sometimes also accompanies respiration; and occurred in Cases VI, VIII, X, XI, and XXV. This frequently cannot be distinguished from that which takes place when the larynx is constricted by inflammatory swelling, and consequently it has occasionally happened that tracheotomy has been performed for the relief of impending suffocation, supposed to have arisen from laryngitis, but which has in fact been caused by the pressure of thoracic aneurism. Case VI, however, proves that an attack of laryngitis may occur in consequence of the irritation produced in the trachea by the pressure of an aneurismal pouch; for not only were the urgent symptoms on one occasion relieved by rapid salivation, but traces of laryngeal inflammation were found after death. When, indeed, the whistling respiratory sound is coupled with an alteration in the position of the trachea, the compression of the trachea by some tumour is made manifest, and in that case it is a valuable sign.

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[1] Lancet, vol. ii, 1840 and 1841, p. 400.

[2] Morgagni de Sedibus Cook, vol. i, p. 454.

When either bronchus is compressed, the only general signs produced are those of cough and dyspnœa, common to so many disorders.

When, however, the substance of the lung is invaded, in addition to these last-mentioned signs, hemoptysis may occur. This may happen, as in Case XXIII, by ulceration between the sac and a cavity or bronchial tube, or by destruction of the walls of the pouch, when the lung itself proves a boundary, as in Cases VIII and XVIII. Hemoptysis took place in Cases VI and IX, from the sac communicating with the trachea. This symptom would, in the generality of cases, induce rather a suspicion of phthisis than of aneurism. Yet in Case IX the nature of the material expectorated, blood and fragments of discoloured coagulum, materially assisted the diagnosis. By the account received it must have been, on one occasion, of the same character in Case XXIII. It will presently be seen that in Case VIII hemoptysis produced a very remarkable alteration in the physical signs, which might have revealed the nature of the affection.

Pressure on the substance of the lungs and its vessels, and the consequent irritation, may produce peculiar effects, as in Cases VII and XXIII, but such a state might, and frequently does arise, without any aneurismal or other tumour being present.

Auscultatory signs—Percussion. Dullness of sound on percussion may concur with other signs to denote the existence of some tumour in the chest; but Case VIII furnishes the only instance in which it proved a valuable sign in conjunction with one general sign alone. The extent of dullness having been considerably diminished after copious hemoptysis, afforded a proof that it was caused by the presence of blood, which could not be conceived to have been diffused in the parenchyma of the lungs, but to have been bounded by an aneurismal sac which communicated with the lungs and bronchial tubes. Extensive aneurismal pouches may exist without giving rise to dullness of sound at the surface of the chest, as in Case XI, and almost in Case X.

Sounds produced by the current of the blood. The manner in which certain sounds are engendered by the current of the blood through aneurismal pouches has been explained. It now only remains to examine their value in respect to diagnosis.

But little can be learnt either from their intensity or quality, because, as has been shown, these properties depend in a great measure on the force of the heart's action. Where we find evidence of the existence of an aneurismal tumour, with little or no murmur, and at the same time we find the action of the heart to be strong, we may conclude that the aneurism is large and sacculated, and that it communicates with the aorta either by a very large or a very small orifice; or else that it is, from some cause, very inelastic; a conclusion, however, which would be drawn from a consideration of the manner in which such sounds are produced, rather than from practical observation. For in all of those cases in which the impulse of the heart was great, some abnormal murmur was heard, except in Case XXI, in which no opportunity for post-mortem examination was afforded. In Case XV the intensity of the murmur was such, actually mounting up into the ear, that, coupled with the existence of an amazingly strong thrill close under the finger, it was taken to indicate the dilatation of a main blood-vessel, so as to make it lie directly under the integuments and sternum. From the situation this could only be the pulmonary artery. In Case XIV the intensity of the murmur decreased with the extension of the aneurismal sac; but in Cases XX and XXXIII it decreased with the diminution of the sac caused by the deposit of coagulum, and the consequent loss of elasticity.

There is one quality of sound which I have never found but in aneurism by dilatation, and that is a hollowness of tone. It was well marked in Cases XI, XV, XVI, XVIII, XXVI, and in the early stage of Case XIII, but subsequently the murmur lost this character.

The seat of the murmur is of great importance. When it is heard at the precordial region it is of much less value than when it is confined to some spot near the upper half of the sternum; because it may then be confounded with a murmur engendered at the aortic orifice, even if it be heard louder near the upper part of the sternum than over the seat of the aortic valves. It may, even under such circumstances, be accompanied with circumscribed dullness on percussion and still not denote the existence of aneurism, because the dullness may be caused by a small amount of solidification of the lung, which, being in contact with the aorta, may transmit the valvular murmur with great intensity to the surface of the chest. This is illustrated in the following case.

Case XXVIII.

Phthisis pulmonalis—Aortic regurgitation.

A merchant, æt. 55, accustomed to free living, began to complain of shortness of breath, which was followed by cough accompanied by clear expectoration, frequently streaked with blood. His strength failing, and losing flesh, he placed himself under Mr. Wickenden's care.

At that time there was dullness on percussion an inch and a half below the right clavicle, and an absence of respiratory sound, and buzzing bronchophony. A double rasp-sound was heard at the precordial region, increasing in intensity until the ear reached the right of the top of the sternum.

Soon after this I saw him and examined him at his request, but not being in attendance on him I took no notes of his case.

On talking the matter over with Mr. Wickenden, I found that latterly he had several attacks of hemoptysis. We considered his case to be one of phthisis; but others considered it might be one of thoracic aneurism.

Soon after this the signs of tubercular softening at the apex of the right lung became very manifest, and he died.

Inspection. The aortic valves were so thickened and shortened that they must have allowed the blood to regurgitate. There was a small dilated aneurism of the abdominal aorta. The lungs were extensively tuberculous.

The murmur was heard both at the precordial region and over the aneurismal tumour in Cases XIII, XIV, XVI, XVIII, and XX.

It was, however, joined by other signs which clearly indicated the existence of aneurism; in Case XIII there being pulsation and absence of pulse in one wrist, in Case XVI pulsation, but the nature of the affection was obscured by the pleuritic effusion. In Cases XIV and XX there was a pulsating tumour; in Case XVIII a pulsating tumour, a purring thrill, and absence of pulse in one wrist.

But even should a murmur be heard confined to a spot near the upper part of the sternum it would not necessarily indicate the presence of aneurism, as is proved by the two following cases.

CASE XXIX.

Constriction of the aorta below the arch—Malformation of the aortic valves—Acute phthisis.

A female servant, æt. 20, had enjoyed good health until three months ago, when, after hard work, she felt pain in the chest, shortness of breath, and headache, which gradually increased, and she became very feverish and thirsty, and was troubled with a dry cough.

On her admission into the Birmingham General Hospital she complained of dyspnœa and inability to lie down, with troublesome dry cough, occasional giddiness, throbbing pains in the head, and slight shooting pains under the left breast. The tongue was dry and brown in the middle, white and moist at the edges. Her appetite was impaired, and she was very thirsty. The skin was dry and hot, the urine plentiful and high-coloured. The bowels were open, but the evacuations were scanty.

Between the second and third ribs on each side of the sternum, and also above the top of it, a well-marked purring thrill was felt. Under the cartilage of the second right rib a loud systolic bellows-sound was heard, gradually diminishing as the ear receded from this spot. It reappeared, but was very faint, between the fifth and sixth left ribs. During the course of her illness the intensity of the bellows-sound varied, but its maximum was always under the cartilage of the second right rib, and near the right sterno-clavicular articulation, where it sometimes amounted to a rasp-sound. The purring thrill also was constant. She died in three weeks from the time of her admission, her chief symptoms having been rapidity of pulse, slight lividity of countenance, great dyspnœa, and hurried respiration, dry cough, with occasional expectoration of clear mucus, now and then tinged with blood, coarse respiratory sound, with cooing sounds, and sometimes muco-crepitant rattles; a combination of symptoms which indicated the existence of unsoftened tubercles or gray granulations throughout a large extent of lung.

Inspection. The arch of the aorta was found to reach as high as the first rib, and was slightly dilated. Just beyond the origin of the left subclavian artery the coats of the aorta suddenly became attenuated round the circumference of the vessel for half an inch, so that in this spot their thickness scarcely exceeded that of an ordinary adult's radial artery; and not only were the walls thinner but the diameter of the vessel was considerably contracted at this spot. At the origin of the aorta were only two semilunar valves, one large and one small, partially adherent to each other, and to the sides of the vessel nearest the mitral valve; so that they formed a funnel-shaped projection into the aorta. They maintained a column of water in the vessel, but offered a slit for the passage of the blood, which only admitted the passage of the little finger. The lungs were densely studded with minute semi-transparent gray granulations, which became rather larger and more opaque towards the apex of each lung.

Case XXX.

Tubercular infiltration of the lung—Constriction of the aorta.

A gentleman, æt. 48, of studious habits, who had scrofulous ulceration of the neck in his youth, complained of a difficulty of breathing which had been coming on for some time. This was followed by a very troublesome cough with slight clear expectoration. He then consulted Mr. Wickenden, who found the whole of the left side of the chest dull on percussion. On this side there was no trace of respiratory sound. On the right side there was coarse respiratory sound, here and there mixed with large and small moist rattles. A loud, single, prolonged rasp-sound was heard over the left side, its maximum being between the second and third costal cartilages. At the precordial region it was heard more faintly, and also in the back. He died, apparently, from increasing dyspnœa.

Inspection. In the right lung there were some gray granulations and unsoftened yellow tubercles, singly and in small groups. The whole of the left lung was converted into a yellowish-white hard mass, of almost fibro-cartilaginous consistence, which was mottled in spots with a pink colour. At the point where the ductus arteriosus joins the aorta the coats of this latter vessel were thickened, and its calibre was reduced to less than half its proper size for nearly the distance of an inch. There was atheroma all along the aorta. The heart was generally hypertrophied and dilated, but not to any great extent. The valves acted well.

In these cases therefore the murmur was caused by constriction of the aorta in the same manner as in Case XXXVIII, where it arose from narrowing of the orifice of the arteria innominata. The purring thrill was perceived in Case XXIX from the arch of the aorta lying much nearer the surface of the chest than usual.

A reference to the manner in which aneurismal murmurs are produced will show that their value as diagnostic signs must in some measure depend upon their connexion with the systole or diastole of the ventricles of the heart. It has been shown that a diastolic valvular murmur can only be formed by aortic regurgitation, or a moderate amount of obstruction at the auriculo-ventricular orifices. In neither of these cases is the murmur carried to such a distance from the spot where it is engendered as when it arises from aortic obstruction. Hence a diastolic murmur, heard *very much* louder on either side of the upper part of the sternum than at the precordial region, is generally, if not always, characteristic of aneurism. Again, if it is heard in such a spot only, it is absolutely characteristic of this disease, because we can suppose no other state of the vessel which could give rise to it. And not only this, but it denotes the existence of sacculated aneurism, the elements for its production not existing in dilatation; and this whether it is accompanied by a systolic murmur or not.

It existed in Cases VIII, XII, XIV, XV, XVI, XVII, XIX, XX, and XXIII, in all of which, except Case VIII, there was a sacculated aneurism. There was, it is true, no post-mortem examination of the body in Cases XII, XVII, and XX; but in Cases XVII and XX there was a pulsating elevation on the surface of the chest, and the blood was at one time felt close under the finger. In Case VIII, however, the diastolic sound was only heard on one occasion a little before death, and might have arisen from some temporary obstruction by a portion of clot. In Case XV the diastolic sound was possibly caused by regurgitation through incomplete valves of the pulmonary artery. The murmur was double in XII, XIV, XV, XVI, and XVII, and single and diastolic in XIX, XX, and XXIII. In Cases XIX and XXIII it was found that the mouth of the sac was so constructed that it would yield to the current of the blood, so that it was allowed freely to enter the sac; but on its exit the calibre of the aorta was diminished, and thereby the current impeded in its onward course. In Case XIV the sound was at first single and systolic, and afterwards double on the aneurism becoming sacculated.

Respiratory sound. There is only one case in which the respiratory sound may be so modified by the existence of an aneurism as to give rise to any sign of its presence; and that is when either bronchus or a main division is compressed by it. This occurred in Cases IX and X. The presence of a tumour was revealed compressing the left bronchus in Case IX, by the total absence of respiratory sound over the whole of the left lung, which sounded clear on percussion. In Case X the respiratory sound ceased in the upper part of the right side on the patient attempting to place himself in an upright or recumbent posture, which showed the existence of a moveable tumour in front of the division of the right bronchus which supplied the upper portion of the lung; and as nothing but an aneurism would satisfy these conditions, this sign was considered characteristic.

From a consideration of the mode of formation and progress of thoracic aneurism, it follows that there is one class in which we cannot expect to find any of the signs which have been shown to possess value as indicative of the nature of the affection in question, and that embraces those aneurisms which arise within the sac of the pericardium. For whilst, on the one hand, they hardly ever reach such a size as to interfere with the contiguous parts, on the other hand, the signs, both general and auscultatory, to which they give rise cannot be distinguished from those which are produced by certain conditions of the heart with which they are in such close contact. Accordingly, in none of the five cases of this kind was the nature of the affection recognised during lifetime, although in two of them auscultation was carefully practised.

The particulars also of two cases of aneurism arising below the bifurcation of the trachea have been given to me, in which the sac was said to have reached a large size without having given rise to any signs by which they could have been recognised before death; but, as I had no opportunity of examining the patients myself, I shall not detail the cases.

A careful analysis, then, of the signs observed in thirty-seven cases of thoracic aneurism leads to the following results.

No diagnostic sign was furnished by the character of the pulse, or by the presence of pulsation above or below the clavicles.

When a pulsation was seen and felt over a prominent spot in the chest, it indicated the presence of a sacculated or mixed aneurism.

Purring thrill was only valuable as a sign of aneurism in conjunction with other signs.

A systolic murmur, heard at a distance from the heart, even though it were not heard at the precordial region, only afforded evidence of the existence of aneurism when it was combined with other signs denoting the existence of a circumscribed tumour.

A double or diastolic murmur confined to one spot, at a distance from the precordial region, denoted the existence of a sacculated aneurism.

When a hollow murmur was heard, a dilated aneurism was present.

The intensity of aneurismal murmur was in a great measure proportioned to the force of the heart's action.

Aneurism of both kinds existed without the slightest trace of pulsation or murmur.

Aneurisms arising within the sac of the pericardium were not indicated during lifetime by any characteristic signs.

I do not bring forward these as general propositions, but merely as results of the observation of thirty-seven cases of thoracic aneurism. Although they are insufficient for the formation of general rules, yet they so far aid the cause of truth as to show that some other writers on this subject have generalized too hastily.

CHAPTER X.

TREATMENT OF THORACIC ANEURISM.

A consideration of the formation and causes of Thoracic Aneurism affords but little prospect of our being able to prevent its approach by prophylactory measures, other than such as are calculated to preserve and improve the general health. For as long as we are ignorant of the special causes which lead to the deposit of atheroma in the arterial coats, on which this disease principally depends, we cannot adopt any special means for its prevention.

When, however, aneurism is once formed, the nature of the treatment which tends to afford relief, and to arrest the progress of the disease, is clearly indicated. The objects to be borne in view are twofold.

1. The prevention of the increase, or rupture of a dilated aneurism.

2. The obliteration of a sacculated aneurism by the deposition of fibrinous clot within it.

In some respects these objects are very similar, in others dissimilar the one from the other. Consequently those writers who have proposed plans of treatment for aneurism generally, irrespective of these differences in the objects to be attained, have failed in adapting their remedies to meet both these cases. I therefore propose considering separately,—the treatment of *dilated*, and of *sacculated* or *mixed* aneurism.

DILATED ANEURISM.

The prevention of further dilatation or rupture may be facilitated by strengthening the walls of the pouch, or by diminishing the force of the current of the blood, or by both means.

The only method by which we can hope to strengthen the walls of the pouch, is by inducing an inflammatory action in them, whereby lymph might be thrown out between the coats. This possibly might be promoted by the use of a highly stimulating quality of food. Such a proceeding would, however, be very hazardous; because, in the first place, it is more than probable that the benefit derived from thus strengthening the walls of the sac, would be more than counterbalanced by other effects of this inflammatory action. Thus the quantity of the general nutrient fluid might be increased, and the exudation into the arterial coats rendered moister, by which means ulcerative softening of the atheromatous deposit might be promoted, and with it the tendency to rupture and dilatation. So also adhesive inflammation, once excited, might be propagated to the contiguous parts, such as the trachea and vena cava, and cause them to adhere to the pouch, and death might thus be produced, as in Cases VI, VII, X, XI, and XXV.

On these grounds, therefore, supposing it possible to induce adhesive inflammation in the walls of the pouch by stimulating food, it would be hazardous to adopt this treatment. Besides which, the heart would also be stimulated, and the force of the current of blood increased; although this might, to a certain extent, be counteracted by measures presently to be detailed.

The balance then being, on the whole, against adhesive inflammation, we are called on rather to prevent it by depletion than to increase it.

The endeavour, therefore, to strengthen the walls of the pouch being abandoned, the only alternative that remains is the diminution of the force of the current of blood.

This may be accomplished by lessening the amount of blood in the circulation, by reducing the heart's action with low diet, sedative and purgative drugs, and mental and bodily repose. In practising venesection, there are two things to be guarded against. One is the danger pointed out by Dr. Watson and Dr. Hope, which arises from a state of irritability which is so commonly associated with weakness, under the influence of which the heart's action would be occasionally more violent than in the ordinary state of health, as was seen in Dr. Hope's experiments on animals. The other is the danger arising from abstracting large quantities of blood, at one bleeding, in the recumbent position, whereby syncope might be induced, from which the heart might not be able to recover, in consequence of the obstacle to the circulation offered by a dilated aneurism of the arch of the aorta full of blood. The same objections lie against an extremely poor diet, and an immoderate use of purgatives, in addition to which such a low state of the system might be induced as would favour the softening of the atheromatous deposit, although in a different manner from that in which such a result is produced in a plethoric state of the body.

In the choice of sedative drugs the danger of syncope must still be borne in view; and therefore the administration of digitalis requires great caution. I have long abandoned its general use in cases of this kind, having found poppy and hyoscyamus much more safe in their action. The extract of belladonna rubbed over the precordial region has appeared to me to act more beneficially in tranquillising the heart's action than any other drug.

In our endeavour to diminish the power with which the blood is impelled into the aorta, we may avoid the danger here pointed out, but still reduce the action of the heart to so low a point, that it will be insufficient to force the blood freely through the capillaries, so that venous engorgement, and its consequent evils, may result. The greatest caution is therefore required in carrying out this plan. With all this, however, it must be admitted that if there be a case in which such a plan can be vigorously pushed with probable advantage, it is that of dilated thoracic aneurism. To the enforcement of mental and bodily repose there cannot be any objection urged, unless it were carried to such an extent as seriously to derange the organs of digestion. The danger arising from a want of it is strikingly seen in Cases XIII, XIV, XVIII, and XIX.

The number of cases to which this treatment is applicable must necessarily be limited, because dilated aneurism is of itself rare, and is seldom detected before the coats have given way, and it has assumed the character of mixed aneurism. Of those here recorded, it was adapted to Cases X and XI, and the early stage of Case XIII. In Case X, however, no great hopes of relief were entertained, from the circumstance that the tumour was already large, and was firmly compressing the trachea. It was not, therefore, pushed to such a degree as to inconvenience the patient. In Case XIII, the patient was only seen occasionally, and could not enjoy rest and quietude.

SACCULATED AND MIXED ANEURISM.

An attempt to strengthen the walls of the sac by the deposition of lymph is not open to the same objections as in the former case; for there is no longer any middle coat to be destroyed by atheromatous softening at that part of the sac which is most prone to burst, nor is the sac any longer bound down to the contiguous parts in such a manner that inflammation could cause it to adhere to them, and so to greatly compress them, because it can expand in other directions; hence there would be no objection against our inducing a moderate amount of adhesive inflammation in the walls of the sac, if we were able to do so, and *a fortiori*, we should not be justified in having recourse to depletion, and lowering, with the view of preventing rupture, or adhesion to contiguous parts.

The obliteration of the sac by the formation of fibrinous clots within it, may be promoted by diminishing the force of the current of blood, in the manner recommended for the treatment of dilatation; because stagnation especially favours the coagulation of the blood. We may by these means, therefore, produce coagulation within the sac, and thus for a time prevent its increase under the expansive force of the current of blood. But a cure may still be very far off. For the sac may be filled with coagulated blood of a very soft consistence, so that not adhering to its walls, the current of blood may pass around it, and burst forth, as in Case XXIV. In order, therefore, that the process of obliteration may go on satisfactorily, it is necessary that the clot should be rich and firm, and become agglutinated to the walls of the sac. Now the copious abstraction of blood, and the restriction of the patient to a low diet, has a strong tendency to impoverish the blood that remains, and to cause it to coagulate into a soft, gelatinous mass, with little tendency to cohesion. Becquerel and Rodier[1] state, that "bleeding exerts a remarkable influence on the composition of the blood, the greater the oftener the bleeding is repeated;" and that in proportion to the number of venesections the blood becomes impoverished and watery. Simon[2] states that in "aneurism arising from excessive loss of blood, the composition of the blood is changed, becoming poor in corpuscles and fibrin; that the solid constituents are diminished; that the clot, if it forms at all, is small, soft, and diffluent, and the fibrin, after it has been separated by whipping, not tough or firm." He also states, that "Gedding, in the examination of the heart and large vessels of anemic inhabitants of the morasses of the Carolinas, found either scarcely any coagulated blood, or else a clear red, or greenish dirty-looking fluid, almost entirely devoid of solid or colouring constituents, containing but few blood-corpuscles, and which could not be coagulated, either by heat or nitric acid." Others, however, do not find the fibrin diminished. In order, therefore, to promote the formation of tough and firm coagula, which may adhere and strengthen the walls of the sac, and on which they may contract, and form a solid fibrinous tumour, we should rather enrich the blood with animal food, than impoverish it by low diet and extensive depletion. This plan having a tendency to strengthen the walls of the sac in the manner explained above, tends also thus to diminish their elasticity, on which the current of blood, in and out of the sac, so much depends, and in this manner greatly favours its coagulation. It behoves us, therefore, to seek for other means to promote coagulation in the sac, which may be compatible with the last-mentioned object.

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[1] Simon's Chemistry. Syd. ed. p. 278.

[2] Op. cit. p. 308.

These may be found in the administration of sedative drugs, purgatives, and chalybeates, mental and bodily rest, and the application of cold to the external tumour.

Were we to trust to rest and sedative drugs alone, whilst we allowed the use of nutritious diet, we should be in danger of inducing a plethoric state of the system. This, however, may be kept under by the use of saline purgatives, which will relieve the system, and remove some of the watery portions of the blood, at the same time that they will not greatly diminish those parts of it which form the large mass of the solid clot. Nor are we excluded entirely from the use of the lancet, which may be occasionally employed with advantage, to withdraw small quantities of blood, so as to relieve any extraordinary tension of the vessels.

The administration of iron has been found by Simon,[1] and by Andral and Gavarret, to have an extraordinary influence on the state of the blood, in some instances doubling the amount of its solid constituents. I have generally given it in the shape of tincture of sesquichloride of iron, with hyoscyamus.

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[1] Simon's Chemistry, Syd. ed. p. 312.

The application of cold cloths and cataplasms, or bladders of ice to the surface of an aneurismal tumour, was practised by Guerin, and subsequently by Pelletan; he joined it, however, to an excessive amount of depletion, and to very low diet. Rejecting the latter part of the treatment, I have been in the habit of applying cold to aneurismal tumours, for the purpose of promoting the coagulation of the blood, and at the same time combining with it the plan of treatment above detailed. In some cases, however, the pain which is induced by the application of cold is so intense, that the remedy cannot be borne. This may sometimes be overcome by using a cold conium poultice.

The rational method, therefore, of treating sacculated and mixed aneurisms, founded on a consideration of their formation, progress, and terminations, would appear to consist in the use of sedative and purgative drugs, a moderate amount of food of a nutritious but not over-stimulating quality, rest of mind and body, and the application of cold to the surface of the chest nearest the sac.

This treatment was attempted in Cases XIII, XIV, XVII, XIX, XX, XXI, XXII, XXIII, and XXIV. In Case XXV there was no time for its trial, and Case XVIII was not under my own care. In Cases XIV and XIX the cold applications could not be borne, from the excruciating pain which they caused. In Case XIII the inability of the patient to afford himself rest from bodily labour prevented him deriving benefit from any treatment. In Case XXV, coagulation in the sac was effected, but the patient was so much lowered and depressed by long suffering and straitened circumstances, that the clot when formed was soft and gelatinous, and thus the blood burst forth before the cure could be effected. The subjects of Cases XVII, XX, and XXI may possibly be still living. In XXII death took place from bronchitis, and the walls of the sac were found solid, and thickened by layers of firm coagulum glued together, and a great part of the sac was filled with similar fibrinous clot, and for some time previous to this attack the patient had attended to his ordinary business. In Case XXIII, death resulted from a communication having been established between the sac and a tubercular cavity. Mr. Fell, in describing the inspection, remarks, "There can, I think, be no doubt entertained as to the effect of your treatment in arresting the tendency to rupture externally, and prolonging her life; and had not the disease been coupled with pulmonary ulceration, affecting the sac (which seemed sound, all around the opening where the fatal hemorrhage took place), it is impossible to say how long she might have lived, or whether she would ultimately have died of aneurism." The morbid parts from both these cases, XXII and XXIII, are in the Museum of King's College, London.

In carrying out this plan of treatment great assistance has been derived from closely watching the nature of the pulsations, gradually discontinuing the means adopted to promote coagulation in the sac, as the decreasing liquidity and clearness of the pulsation indicate its progress. Deducting Case XIII, in which the circumstances in which the patient was placed prevented his allowing the treatment to be carried out, we have, out of eight cases, five in which great relief was obtained, and three in which the treatment failed.

Thus, as far as the limited number of cases allows of any inference being drawn, practical observation confirms the soundness of these views of treatment, which are suggested by the nature and progress of the affection.

Success, however, has been claimed for a line of treatment, in some respects the opposite of that here recommended, viz. the depletory plan as practised by Valsalva. Dr. Hope has pointed out the serious objections which exist against this treatment, and has proposed a plan more approaching to that which is here advocated. Drs. Beatty, Stokes, Watson, Copland, O'Brien, and others, have also advanced sound reasons against our employing Valsalva's method. The extreme severity of this treatment would prevent its being in most cases rigorously carried out, even were there not so many weighty reasons against it; and hence also it is probable, that in many cases the cures claimed for it have in fact taken place under the use of a diet rather more generous than was suspected by the medical attendant.

Now, in selecting the treatment to be adopted, as we cannot always determine whether an aneurism is dilated or sacculated, it is a safe rule to adopt the treatment for dilated aneurism, in those cases in which the tumour has not reached the surface of the chest, and the treatment for sacculated aneurism in those in which an external prominence is perceived. Occasionally, but rarely, a dilated aneurism would be included in the latter class, as Case XVIII, but never one in which the coats were entirely preserved; sometimes also a sacculated aneurism might be included in the former class (Case XIV in its early stage); but even if this were so, the danger of adhesion to an important contiguous part, as the trachea, would be diminished by the antiphlogistic treatment, and on the sac reaching the surface, the treatment would be immediately changed.

CHAPTER XI.

PROGRESS AND TERMINATIONS OF CHRONIC HEART DISEASES.

There is a large class of cases in which the health is slowly impaired, and death eventually induced by derangements of the circulation, which depend on organic changes of the heart. These changes affect its contractile power and its valvular apparatus, just as the work performed by an engine is proportionate to its moving force, and the state of its machinery.

The principal of these organic lesions are hypertrophy, attenuation, and structural alteration of the muscular walls of the heart, on which its contractile powers depend; and valvular derangements, which either interfere with the perfect closure of the different orifices of the heart, and thereby permit a regurgitation of the blood, or else offer obstacles to the onward flowing of the blood in its normal direction.

It is seldom that any one of these changes is alone concerned from first to last in producing derangements of the heart's action. Oftener several of them are found in combination, and not unfrequently some produce effects antagonistical to those which result from others. Thus when hypertrophy of the left ventricle coexists with narrowing of the aortic orifice, the increased velocity of the blood compensates for the diminished size of the stream which enters the aorta.

Hence something more is required for practical purposes than the separate study of each of these organic lesions, and of the effects they have on the motion of the blood. It is proposed therefore to examine the manner in which the health is impaired by the various derangements of the heart's action thus induced, and the steps by which a fatal termination is approached.

Death may result from a direct stoppage of the action of the heart, or from impediments offered to one or both of the circulations which result from it.

Its motions may be directly arrested by syncope, inflammation, or various accidents, in a manner which requires no explanation or comment; but it is not easy to explain, in every case, the mode in which one or other of the two circulations are impeded.

Some persons assert that arterial congestions, followed by fatal consequences, are produced in the different organs of the body by hypertrophy of the ventricles, the enlargement of the right giving rise to pulmonary apoplexy, hemoptysis, &c.; and that of the left ventricle to cerebral apoplexy, engorgement of various viscera, and to general dropsy. But the main cause of impeded circulation is supposed to be disease of the orifices of the left side of the heart, acting in the first instance on the pulmonary circulation. The vessels of the lungs being thus engorged, are supposed to be incapable of freely receiving the contents of the right ventricle, which being unable to empty itself, can no longer receive the usual quantity of blood from the right auricle, and thus this fluid stagnates in the veins, and the general circulation becomes also impeded. It is not denied that the latter effect may be directly produced by alterations of the orifices and valves of the right side of the heart; but such derangements are supposed to be so rare, that they very seldom are the causes of impeded general circulation.

Dr. Hope, however, considers dilatation of the heart to be a more direct and efficient cause of impeded general circulation than valvular disease. He remarks, "I have repeatedly witnessed cases in which a well-marked, if not a considerable obstacle, as a contracted valve, a regurgitation, or a dilatation or aneurism of the aorta, had existed for a long period, even for years, without producing any material symptoms of an obstructed circulation; but the moment that dilatation of the heart supervened, the symptoms made their appearance in an aggravated form."[1]

The truth of this remark will be attested by all those who have had extensive opportunities of examining this class of diseases. Dr. Hope supposes the attenuation and consequent weakness of the walls of the ventricles, whether dilated by the pressure of obstructed blood, or by the debilitating effects of fever, chlorosis, inflammation, &c. &c., to be the cause of the engorgement of the general circulation. This explanation does not apply to those cases of frequent occurrence, in which hypertrophy is joined with dilatation. Andral endeavours to meet this case by the supposition that the impediment "arises from the excess of the capacity of the heart relative to that which has been preserved in the blood-vessels."[1] Cases, however, in which excessive dilatation and hypertrophy existed for many years without giving rise to any symptoms of impeded circulation, will be presently adduced.

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[1] Diseases of the Heart, 3d edit. p. 301.

It is probable, therefore, that there is either some cause of obstruction connected with dilatation as yet undiscovered, or that one of its known causes has in many cases been overlooked; and as the treatment adapted to ward off fatal terminations must in some degree depend on a knowledge of the steps by which they are reached, it is of great practical importance to ascertain correctly the manner in which these steps are produced, and the order in which they succeed to each other.

With this view it is proposed to examine a certain number of cases of diseased heart, which were recognised as such during lifetime, distinguishing those which were accompanied by symptoms of obstruction of the general circulation from those in which no such symptoms occurred, in order to ascertain the conditions under which these different results take place.

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[1] Clinique Médicale, Brussels, 1830, p. 104.

CLASS I.

CASES OF HEART DISEASE EXHIBITING NO TRACES OF OBSTRUCTION OF THE GENERAL CIRCULATION.

Case XXXI.

Aortic and mitral regurgitation.

A gun-barrel filer, æt. 30, the notes of whose previous history are mislaid, at the time of his admission into the Birmingham General Hospital was emaciated, and laboured under great dyspnœa. All the arteries which approached the surface were seen to pulsate violently. There was a strong heaving impulse two inches below the left nipple. The pulse was resilient. Above the nipple and up the course of the aorta there was a double rasp-sound, the diastolic being stronger, coarser, and more prolonged than the systolic sound. Two inches below the left nipple the systolic sound alone was heard. Fourteen days after this a purring thrill was felt up the course of the right carotid, but not up the left. During the act of coughing a lump as large as a nut was seen to rise up above the right clavicle, over which a purring thrill was felt. In that spot a single systolic rasp-sound was heard. To the left of the sternum the diastolic sound was faintly heard, but under the middle and bottom of the sternum it was rough and prolonged.

No more notes of this case during lifetime can be found. He died five weeks after his admission into the hospital.

Inspection. The pericardium adhered to the side of the thorax on a level with the sixth rib. It contained five ounces of clear lemon-coloured serum. There were milky spots on the heart, which was firm, red, and rather small. The wall of the left ventricle was eight lines thick across the middle. The aortic valves were thick, shrivelled, and small, with cartilaginous bases; they could unfold, but were insufficient to close the aortic orifice. The circumference of the mitral orifice measured four inches. The ventricular side of the mitral valve nearest the aorta was covered with patches of warty vegetations, and was eight lines in height. The other valve was thick, shrivelled, and much shorter, and all the cordæ tendineæ were shortened and thickened. Two of the tricuspid valves were one inch deep, and the other half an inch. The circumference of the tricuspid orifice measured three inches and a half. The arteria innominata was contracted at its origin and dilated beyond that point. There were some patches of old adhesion between the bottom of the left pulmonary pleura and the side. The top of the left lung was surrounded by a cartilaginous cap, and the lung itself at this spot was puckered and contracted. Both lungs were much engorged, and there were patches of brown-coloured, dryish hepatization, in which there was no crepitation, nor was there any offensive odour. There was no trace of dropsy.

The notes of a clinical lecture on this case, which have been preserved, show that the leading symptoms throughout were dyspnœa and palpitation, and that death ensued from what may be styled chronic suffocation, without a trace of anasarca having been manifested. A comparison between the physical signs during lifetime and the appearances after death is remarkably instructive.

Case XXXII.

Mitral regurgitation —Diseased bronchial glands —Pulmonary emphysema.

A lad, æt. 19, of diminutive stature, not looking more than twelve years of age, stated that he had never been put to any kind of work, having been, from a child, unable to bear exertion; in youth because his body was covered with an eruption, which, about five years ago, was cured, soon after which his breathing became very short.

On his admission into the Birmingham General Hospital there was slight spinal curvature to the left perceived. The chest was bombed and rounded by the filling up of many of the intercostal spaces. The respiration was laborious and chiefly abdominal. His face was livid and bloated. He complained greatly of dyspnœa, amounting to a sense of suffocation, on endeavouring to walk quickly, and of a troublesome cough and wheezing.

Percussion everywhere elicited a clear sound, but more especially under the sternum and behind at the lower third of the left side, where it was ringing. The pulmonary sound, everywhere feeble, was absent in certain parts, particularly under the sternum. In front it was accompanied by varieties of moist rattles; behind the lower two thirds of each lung there was a tolerably even and fine muco-crepitant rattle. The voice resounded more clearly than usual under the sternum and behind the left side. The sounds of the heart were natural.

This lad improved considerably after having been leeched between the shoulders, and after having taken the *mist. ammoniaci* with mild laxative medicines. Nine days subsequent to his admission he was seized with great dyspnœa after exerting himself in walking, and died in two hours.

Inspection. On opening the chest the lungs did not collapse. Nearly the whole of the free edge of each lung was studded with patches of emphysema, the cells of which were not larger than mustard seeds; similar patches existed at different parts of the surface of the lungs, especially behind the left lung. The bronchial glands were much enlarged, and rose up into the anterior mediastinum, varying in size from that of a pea to a pigeon's egg. They resembled the spleen in colour and consistence. In one near the root of the lungs was an atheromatous yellow mass as large as a horse-bean, of the consistence of chalk. Much blood and serum ran out on cutting into the lungs, the lower lobes of which were greatly engorged. The mucous membrane of some of the air-tubes was thick, soft, and violet-coloured. The heart was equal to that of a full-sized person, but the circumference of the aorta did not exceed that of the forefinger. The heart was of a yellowish-brown colour and flabby. The edge of one of the mitral valves was divided into three half-lozenge-shaped points, having a great many very short cordæ tendineæ, with little or no carneæ columnæ. The cords were so short, and the valve had so little depth, that it could not rise up into the plane of closure. The mitral orifice was four inches and a half in circumference.

The origin of deranged circulation may here be traced to congenital malformation of one of the mitral valves. On the enlargement of the cavities of the heart by natural growth, whilst this shallow valve remained tied down, a certain amount of regurgitation must have taken place through the mitral orifice. This would have a tendency to increase from a still further dilatation of the orifice itself, produced by the pressure of the blood from the lungs, and the back current during the systole of the ventricles. A diminished supply of blood being thus poured into the aorta it would cease to grow by a well-known law, thus accommodating its calibre to the amount of duty required of it. The body, also, would not grow with its usual vigour in consequence of receiving a deficient supply of arterial blood, and hence this lad's stunted appearance. That he should have reached nineteen years of age is only to be accounted for by his never having been put to any employment.

Case XXXIII.

Adherent pericardium—Mitral and pulmonary regurgitation.

A gas-burner maker, æt. 30, a confirmed drunkard, after taking cold was ill three months, and ever since has had a pain at the heart, and one night whilst smoking felt as if something had burst there. Since then he has had more or less of palpitation, but has continued to work. Three months since he got wet through, and began to cough and expectorate white phlegm; the pain at the heart became severe, and he had also pain in the epigastric region. Palpitation increased, he lost flesh and strength.

He was bled and blistered without much relief. On his admission into the Birmingham General Hospital he had strong palpitation, and complained of violent pain at the heart on the least exertion. He had slight cough with clear expectoration, and great dyspnœa on moving. He was much troubled with dreams. His bowels were confined, and his urine was scanty and high-coloured, and deposited a sediment like bran, but contained no albumen. The pulse was 116, feeble.

The chest sounded nowhere very clear on percussion, but was very dull from the sternum to the outer edge of the left side below the left nipple. The respiratory sound was weak over the front of the left side of the chest. The action of the heart was very strong and quick, like the stroke of a hammer.

The sounds were sharp, and were heard all over the chest, the diastolic sound being a short bellows-sound, which was afterwards occasionally absent. A metallic resonance accompanied the heart's impulse. He soon died without any trace of dropsy, and apparently from dyspnœa.

Inspection. The lungs were congested but not friable. The pericardium adhered to the whole of the heart by means of a gelatinous lemon-coloured substance. The heart was placed almost transversely across the chest, its apex reaching to the extreme left side of it, and the whole of this space was uncovered by lung. It was greatly dilated, more particularly on the left side. The parietes of both auricles were hypertrophied, especially the left, which in one place were four lines in thickness. The lining membrane of the left auricle was opaque and thickened, having horny patches on it, but inseparable from it. The mitral orifice was a little contracted, so as with difficulty to admit two fingers. The free edges of its valves were covered with fibro-cartilaginous vegetations, of the consistence and appearance of soft horn; two of them being three quarters of an inch in length, five lines wide, and two lines thick. These bodies were fringed with vermiform vegetations, about the size of crow-quills. The valves were thickened and large, but seemed adapted to each other, the vegetations hanging into the ventricle when they were closed. The orifice of the pulmonary artery was so much dilated that it admitted four fingers.

The stomach was pushed downwards by an immense liver and spleen. The liver was hard, and had a mottled nutmeg appearance. The spleen measured eleven inches by six. Its upper half was reduced to a dirty red pulp, and adhered to the diaphragm. The edge of the softened part was coated with yellow cheesy-looking matter; of which considerable patches existed in the remaining half of the spleen. The cortical substance of the kidneys was pale.

This man was examined by permission of Dr. James Johnstone, and the notes are published with his knowledge and approval.

He would appear to have had repeated attacks both of pericarditis and endocarditis; the gelatinous substance which connected the pericardium being the product of the last recent attack. The enormous vegetations on the mitral valves must have materially interfered with their action, and probably sometimes got between them, and thus allowed regurgitation to take place. Had the diastolic bellows-sound been occasioned by regurgitation through the pulmonary orifice, it would have been constantly heard. The space allowed for regurgitation was probably too large to give rise to sound. It is more likely that this sound arose from obstacles to the flowing of the blood through the mitral orifice, varying with the position of the large horny excrescences.

Mitral regurgitation can have little effect in obstructing the general circulation, otherwise more decided symptoms of such an obstruction would have showed themselves in this case. Case XXXI also illustrates this.

Death probably resulted from the joint effects of pericarditis and mitral obstruction.

Case XXXIV.

Mitral and aortic obstruction—Bronchitis.

A widow, æt. 50, had always laboured under delicate health, and for some years had experienced dyspnœa on exertion. Latterly this had much increased, and was followed by pain in the head and sickness, and then by palpitations and occasional diarrhœa. When visited in consultation with Dr. Skerrett, she had a troublesome cough with muco-purulent expectoration. There was some œdema of the ankles. The tongue was clean and the bowels regular. The urine was turbid, and not coagulable by heat. There was no pulse perceptible at the right wrist, in the left it was frequent.

The precordial dullness was extensive. On the right side of the chest there was a mixture of cooing sounds, and slightly ringing mucous rattle. The sounds of the heart were distant and confused; the diastolic sound having none of its usual clearness. There were no venous pulsations in the neck. In a few days' time she died from increasing dyspnœa.

Inspection. Both lungs were universally adherent to the chest. They were much congested, and the lining membrane of the bronchial tubes on the right side was in many places swollen, livid, and softened; they contained much muco-purulent fluid.

The heart was dilated, particularly on the right side, but not hypertrophied. The tricuspid valves, however, were large, and supported fluid, although the orifice was upwards of five inches in circumference. The mitral valves were fibro-cartilaginous, and their bases adhered to each other, so that one finger only could be introduced between them. On the auricular side of one of them was a softish mass of semi-organized fibro-cartilage. On the lining membrane of the auricle were old patches of white lymph, and also recent bloody patches. The bases of the aortic valves were ossified, and numerous patches of atheroma were found in the aorta. The arteria innominata was nearly obliterated by similar deposits in its middle coat, and between it and the lining membrane.

The kidneys were yellowish, and slightly lobulated and mottled.

The right side of the heart being considerably dilated in this case, and with it the tricuspid orifice, from the naturally small size of the tricuspid valves, it might have been expected that a portion of the dilated orifice would have been left uncovered by them; they were, however, unusually large in this case, and effectually closed the tricuspid foramen.

Death was produced by bronchitis occurring in lungs previously congested by mitral obstruction.

Case XXXV.

Adherent pericardium—Mitral obstruction.

A dress-maker, æt. 29, single, had rheumatic fever, attended with violent pain in the left side, at the age of 17, and ever since that time has had palpitations and dyspnœa, which, on exerting herself, were most urgent and distressing. She was also subject to a teasing cough, with clear mucous expectoration.

Outside the left nipple, between the seventh and eighth ribs, undulations were seen, corresponding with the impulse of the heart. Some cooing and bass-viol sounds, with traces of fine muco-crepitating rattles, were detected in different parts of the chest. A shrill rasp-sound was heard with the systole of the heart; being at its maximum under the left nipple, and at the angle of the left scapula. She was relieved by sedative and expectorant medicines, and by belladonna frictions over the heart. Soon afterwards she had an attack of left pleurisy,—this was at the age of 27. Two years after this she was suddenly seized with a violent pain under the left nipple. Her pulse was sharp, small, and frequent, 120. The eyes and face were injected.

There was no dullness on percussion. The respiratory sound was natural, but there was a creaking at the outer part of the left side. A systolic and rather prolonged bellows-sound was heard at the left nipple, but did not extend far from it. She soon became delirious, and died in a few days.

Inspection. The outer and lower part of the left lung was connected with the side by bunches of white threadlike cords, from one to four inches in length. The free edge of this lung adhered to the pericardium. The lungs were healthy, but much congested.

The pericardium was closely and universally adherent to the heart, and also to the chest at the seventh and eighth ribs, by means of bands similar to those which connected the pleural surfaces. The heart was of a natural size. The mitral valves were thickened, and at the base of one of them was a patch of fibro-cartilaginous matter, as large and as thick as a bean. The lining membrane of the left auricle was of a deep violet colour, and was rough and granulated with whitish raised grains, over a space as large as a crown-piece, running into the mitral orifice.

The history of this case renders it probable that the adhesion of the pericardium took place at the period of the first attack, when this patient was seventeen years of age. The bands which connected it with the outer side of the chest were certainly not of recent formation; and the undulations in the side to which the adhesion gave rise had been seen two or three years before her death. The uneven thickened edges of the mitral valves must, for some time, have prevented their perfectly closing the mitral orifice, and hence there was regurgitation through a narrow slit, giving rise to sharp, shrill systolic sound. The fatal termination was hastened by an attack of endocarditis.

Case XXXVI.

Aortic and mitral obstruction—Sudden death from syncope.

A tradesman's wife, of small stature and delicate constitution, a native of Poland, had rheumatic fever at six years of age. Latterly she had suffered from palpitations and dyspnœa on over-exertion or excitement. During the last week she had felt better than usual, and was, with her husband, spending the evening with some friends in a small room with a large fire in it, where she appeared very happy, and not excited. Having taken a little bread and cheese and half a glass of ale for supper, she complained of the heat, and removed further from the fire. Presently she suddenly threw out her right hand as if to grasp something, turned deadly pale, and dropped her head on her husband's shoulder. I saw her in less than ten minutes, supported in an upright position, and quite dead.

Inspection. The vessels of the brain were almost empty. The upper third of each lung adhered to the thorax, and both were engorged with blood, except at their very summits. The heart was double its natural size, from hypertrophy and dilatation of its left side. The bases of the mitral valves adhered to each other in such a manner as to allow the passage of only one finger. The lining membrane of the left auricle was opaque, and partially thickened. The aortic valves were much ossified, and one rigidly projected from the side and could not lie flat.

Death was directly produced in this instance by syncope injudiciously managed; but the patient had a short time before her death been under my treatment for palpitation and dyspnœa on exertion. No abnormal sounds of the heart were then detected.

It is instructive to observe how much disease may exist on the left side of the heart without giving rise to urgent symptoms. Here was both aortic and mitral obstruction.

Case XXXVII.

Slight aortic obstruction—Hypertrophy and dilatation—Sudden death.

A young man, of delicate constitution and slender make, æt. 19, had never enjoyed such an amount of health as would allow him to follow any employment. He had experienced strong palpitations at the heart from his earliest recollection, which were much increased by exertion, and were attended with a pain and throbbing up his neck to his head. He had generally some cough, with slight and clear expectoration. On one occasion he spat up three or four table-spoonfuls of blood after walking quickly. The pulse was full, hard, and resilient. The action of the heart was heaving, and was seen all over the chest, and all the arteries near the surface were observed to pulsate violently. A strong pulsation was felt over each clavicle, and over the sternum on pressing down the finger. A thrill was also felt up the right carotid, over the right clavicle, and behind the sternum on deep pressure. There was extensive dullness around the region of the heart. The cardiac sounds were distant and confused when listened to over the precordial region. At the top of the aorta, and up the right carotid, was heard a double rasp-sound. The respiratory sound was natural.

He was seen occasionally both by Mr. Alfred Baker and myself until the time of his death, which took place suddenly rather more than three years after this. He had been sleeping in his chair, when he awoke and suddenly placed his hand on his heart. He dropped his head and died, his sister declaring she heard a noise at his heart.

Inspection. The body was opened under a coroner's warrant, much against the wish of his friends, and to satisfy them a promise was given that the chest only should be examined.

There was some little serum in both pleural cavities, more in the left than the right. The bottom of the left lung was carnified. The pericardium contained between three and four ounces of serum. The heart had some large milky patches on it, and was of enormous size, the bases of its ventricles measuring fifteen inches and a half round, and the left ventricle nine inches from its apex to its base. The walls were firm and red. The tricuspid orifice was five inches in circumference, and its valves were very large and sufficient to close it. A mass of calcareous matter adhered to the base of one of the sigmoid valves, not quite so large as a small horse-bean.

The heart in this case had no appearance of disease, but seemed rather the healthy heart of a giant. There is every reason to believe that it was unnaturally large from birth.

Here were the conditions which have been supposed by Andral to be peculiarly favorable to the production of serous effusion: viz. great disproportion between the size of the cavities of the heart, and that of the aorta; and yet no other evidences existed of obstruction of the general circulation than were furnished by the presence of a little serum in the pericardium and thorax. Aneurism of the arch of the aorta was at one time suspected.

CASE XXXVIII.

Hypertrophy and dilatation of the heart—Large tricuspid valves—Diseased arteries—Sudden death.

A married woman, æt. 50, had a severe illness eight years ago, with pain in her left side, since which time she has occasionally felt pulsations extending from her heart up to her head, and down her arms, with giddiness. She has had latterly a constant cough, and when examined the pulse was small. Pulsations were felt above each clavicle. The action of the heart was heaving and tumultuous. The carotid arteries pulsated very strongly. Double saw-sound was heard, its maximum being under the bottom of the sternum. A systolic saw-sound was heard under each clavicle, loudest under the right, without any diastolic sound.

Two years after this, being excited, she suddenly placed her hand over her heart, drooped her head, and died in half an hour.

Inspection. The heart was very much increased in size, being generally hypertrophied and dilated, more especially the left ventricle. The tricuspid foramen measured five inches in circumference; but the valves were hypertrophied in substance and extent, so that they effected the closure of the foramen perfectly. The aortic valves were slightly cartilaginous, but acted well. The lining membrane of all the arteries, particularly the aorta, was much diseased, being puckered and having much atheroma under it, and being deficient in some places. The arteries of the arms were diminished in calibre by the deposition of atheroma in their middle coats. The other organs were healthy.

Here also was considerable general hypertrophy and dilatation, without any symptoms of obstruction of the general circulation.

In this case aneurism of the arteria innominata was at one time suspected. The double saw-sound was not accounted for by any post-mortem appearances.

Case XXXIX.

Bronchitis and emphysema—Effusion in the chest—Dilated tricuspid orifice, with large valves.

A labourer, æt. 60, had been subject to cough and shortness of breath two winters, and had been in bad health for twelve months, during which time his feet and ankles had occasionally swelled. On his admission into the Birmingham General Hospital his lips were livid and his face bloated. He laboured under excessive dyspnœa, with orthopnœa. He had cough, attended with scanty muco-purulent expectoration. The skin was cool, the pulse not frequent, but firm and quick. The bowels were regular. The urine was scanty and loaded with lithates, but contained no albumen. He had occasional palpitations and considerable œdema of the feet and ankles. The external jugular veins were tumid, and slight undulations were seen in them. The precordial dullness was much more extended than usual; and the chest was bombed out over this spot. The pulmonary sound was masked by loud cooing sounds and mucous rattles of different kinds. The action of the heart was slow and heaving. Its sounds were limited to the precordial region, and were distant and muffled. Ten days after this his urine was increased, and the œdema of the legs was nearly gone, from his having kept a horizontal position and having taken diuretic medicines. But dyspnœa had greatly increased. He lay on his left side, with his head raised, not being able to lie on his back or right side without a sensation of suffocation. His face was very much puffed and very livid; his cough frequent and dry. The left side sounded dull below the fifth rib, varying with position, and here only an obscure gurgle was heard on deep inspiration, but there was no other respiratory sound. There was bleating resonance of the voice along the margin of the fifth rib. Above this rib, on the right side, the pulmonary sound was coarse, and mixed with mucous and muco-crepitant rattles. The impulse of the heart was very feeble, and the sounds distant and prolonged. He died from dyspnœa in four days more.

Inspection. The left pleural cavity contained a pint and a half of serum, and the right half a pint. The free edges of both lungs were studded with patches of emphysema, and their posterior parts were congested. The left lung was somewhat dense and compressed. The lining membrane of the bronchial tubes was livid, softened, and thickened irregularly. The pericardium contained ten ounces of serum. The heart was large, and weighed twenty-eight ounces. All its cavities were somewhat dilated. The walls of both ventricles and the carneæ columnæ were slightly hypertrophied. There was much atheroma in the aorta. The aortic valves were a little thickened at their bases. The tricuspid foramen measured five inches and three quarters in circumference, but the valves were unusually large, being on an average nearly one inch in height. There was no serum in the abdomen.

The first and principal diseases in this case were doubtless bronchitis and emphysema of the lungs, and the pulmonary congestion attending them was the cause of the dilatation of the right side of the heart. As the tricuspid valves were uncommonly large, there would be no regurgitation, but yet there were undulations of the jugular veins.

Case XL.

Mitral contraction—Pulmonary congestion.

A gentleman, æt. 36, had been ill some time, suffering chiefly from dyspepsia and dyspnœa. Shortly before his death he was seen by Mr. Saunders, when the dyspnœa was very distressing. Hemoptysis of dark coloured blood succeeded, and he died suffocated, having never exhibited any signs of anasarca.

Inspection. The heart was large, being dilated and hypertrophied; and there was such an amount of calcareous deposit around the mitral orifice as to reduce it to a very narrow slit. The lungs were amazingly congested with dark blood, and some patches of pulmonary apoplexy were found in them.

Case XLI.

Mitral obstruction—Pulmonary and cerebral apoplexy.

A female, æt. 39, was in good health when she ran violently with her breast against a post. From that time she felt a pain in her chest, and her breathing became short. Whilst under the care of Mr. Saunders, she had several attacks, both of cerebral apoplexy and of hemoptysis. In one of these attacks of apoplexy she died, having never had any trace of anasarca from first to last.

Inspection. The lungs were studded with large patches of pulmonary apoplexy. The heart was large, and the mitral orifice was excessively contracted by fibro-cartilaginous and calcareous deposits, so that the little finger could not pass through it.

The last two cases were furnished by Mr. Saunders, who kindly presented me with the preparations.

They prove that diseases of the valves of the left side of the heart may proceed to a great length without inducing the slightest trace of obstruction of the general venous circulation. It is probable, however, that congestion of the veins of the general circulation had occurred in Case xli, and that the fatal attack of apoplexy was the result of it. The state of the tricuspid orifice, however, was not noted.

CLASS II.

CASES OF DISEASED HEART, WITH SYMPTOMS OF OBSTRUCTION OF THE GENERAL CIRCULATION.

Case XLII.

Mitral, aortic, and tricuspid regurgitation—Dropsy—Effusion on the brain.

A filer, æt. 38, was a patient of the Birmingham General Hospital.

All the notes are mislaid except those of the inspection of the body, and of a clinical lecture delivered on his case. From these it appears that his earliest and leading symptoms were dyspnœa and great pain at the precordial region. His pulse was resilient and small, and was visible at the wrists. A double rasp-sound was heard, of which the systolic portion was very rough, and very much prolonged, and was at its maximum below the nipple, neither of the sounds extending up the course of the aorta. Subsequently, venous pulsations of the neck appeared, followed by œdema of the feet, which rapidly gained the thighs, scrotum, and abdomen. While on the night-stool he became suddenly comatose, and died soon after.

Inspection. The vessels of the brain were much congested, and there were four ounces of serum in its ventricles. There was a little serum in the right pleural cavity, and much more in the left. The right lung was congested, the left very much so, and its posterior portion adhered to the chest.

The pericardium was distended with straw-coloured serum. The heart was greatly dilated, and the walls of the left ventricle much hypertrophied. The mitral orifice was nearly obliterated by calcareous deposits, and its valves were short and cartilaginous, not free to act. The aortic valves were short and corrugated. The tricuspid orifice measured nearly six inches round; the valves were thickened. The aorta was dilated, and roughened by atheroma. The liver was large and congested.

The mitral disease was of long standing, but, formidable as it was, no symptom of obstruction of the general circulation appeared, till venous pulsations in the neck announced the accession of tricuspid regurgitation, which arose from the inability of the valves to close the greatly dilated orifice.

Case XLIII.

Pericarditis—Mitral contraction—Tricuspid dilatation—Dropsy.

An iron-caster, æt. 26, had an attack of rheumatism between six and seven years ago, from which he quite recovered. Eighteen months since he first expectorated blood, after carrying a heavy load, and has occasionally done so ever since, under similar circumstances. He has been in the habit of drinking much ale. Sixteen months since he caught cold, and suffered from severe pains about his left nipple, cough, dyspnœa, and palpitation. These symptoms have more or less persisted, being all aggravated by exertion.

On admission into the Birmingham General Hospital he was very pale; his feet and ankles were slightly œdematous, the tongue was of a natural colour, rather excoriated in spots. His pulse was 104, small and quick. He complained of great thirst, and pain under the left nipple shooting backwards, occasionally with great severity. He had cough, with expectoration of clear mucus, and occasional vomiting. Palpitation and dyspnœa were both distressing on exertion.

A pulsation was seen to the left of the xiphoid cartilage, and a purring thrill was felt below the left nipple and there alone, and the veins of the neck were turgid. The diastolic sound of the heart was strong and sharp. A double rubbing sound was heard below the left nipple, diminishing in intensity with the distance from this point in every direction. He improved slightly for nearly a month, under tonic and expectorant medicines, and anodyne frictions over the heart, when the cardiac pain became rather suddenly aggravated, and with it dyspnœa, palpitation, and cough. The legs and scrotum became rapidly anasarcous. The pulse soon reached 140, and was irregular. The veins of the neck undulated. The expression of the countenance became most anxious. The action of the heart was fluttering and tumultuous. Its sounds were masked by a double rubbing sound close to the ear. The precordial dullness was extended. Mercury was actively rubbed in over the heart. But, in spite of all treatment, he sank in a week's time.

Inspection. The right lung adhered firmly to the chest. Both lungs were engorged, but healthy in structure. The outer surface of the pericardium was congested and thickened, and on it were several patches of rough lymph. It contained 12 oz. of straw-coloured serum. Its internal surface was highly injected, and its posterior portion adhered to the heart, and on the other part there were rough patches of lymph, and also on the corresponding parts of the heart.

The heart was enlarged by dilatation of its right side, which contained a large coagulum. The tricuspid orifice was six inches in circumference, the valves were thickened. The mitral ring was cartilaginous and calcareous, as also were the valves, so that the point of the little finger could not pass through it. The calibre of the aorta was small. The coronary vessels were dilated.

The liver was large and congested. The kidneys were also congested, and appeared slightly granular.

Two distinct attacks of pericarditis may be clearly traced in this case, the second of which hurried on death, both by directly interfering with the heart's action, and also by producing softening of its walls, and thereby favoring their dilatation. For by this latter means regurgitation through the tricuspid orifice, which must already have taken place from the corrugation of its valves, would be greatly increased when the circumference of the orifice was dilated to six inches.

The progress of the disease is well marked; at first the symptoms of pulmonary congestion appearing in consequence of mitral obstruction, and then those of impeded general circulation, as the right side became dilated, and with it the tricuspid orifice.

CASE XLIV.

Adherent pericardium—Mitral obstruction—Tricuspid regurgitation—Dropsy.

A widow, æt. 55, ever since the catamenia disappeared, five years since, had felt dyspnœa, palpitations on exertion, with cough, and expectoration of clear, grayish, lumpy mucus. Three weeks since she took cold, when her cough became aggravated, and she felt a pain at her heart; the dyspnœa and palpitations became worse. She was unable to lie down for a week, and her legs had swelled, and pitted on pressure for four days. Her urine was scanty.

When visited her pulse was small, irregular, and feeble. There were large undulations visible in the veins of the neck, which were turgid, particularly those on the right side. The dullness over the precordial region was extended. The pulmonary sound was accompanied by bass-viol and cooing sounds, and some mucous rattles. The action of the heart was very tumultuous, irregular, and feeble. The sounds were extended and weak, but natural.

She improved for a time under a generous diet, belladonna frictions over the heart, and tonic and diuretic medicines. But relapsing, she entered the Queen's Hospital. Finding herself getting worse, she again went home.

On being summoned to her I found her deeply jaundiced, labouring under extensive anasarca, and in a dying state.

Inspection. The lungs were engorged, and there were several large masses of pulmonary apoplexy, with well-defined edges.

The pericardium adhered closely to the heart. The heart was large, yellow-mottled, flabby, and soft. The auricles were greatly dilated. The tricuspid orifice measured four inches and a half in circumference, its valves were opaque and thickened, and had very short shrivelled cordæ tendineæ. The mitral ring was osseo-cartilaginous, and contracted so as only to admit one finger, the valves were cartilaginous, but would close the orifice.

The liver was rather large, its substance bordering on cirrhosis. It adhered to the abdominal walls.

The progress of disease was well marked in this case also. Obstruction at the mitral valve and shortening of the cords of the tricuspid valves had probably existed for some time; but the pericarditis which led to adhesion having set in about three weeks before the patient was seen, caused a rapid dilatation of the tricuspid orifice, and thus a formidable regurgitation would take place.

Case XLV.

Adherent pericardium—Mitral and tricuspid regurgitation—Emphysema of the lungs—Dropsy.

A brassfounder, æt. 35, of temperate habits, had a cough, with clear frothy expectoration, as long as he could remember, with dyspnœa, which was aggravated every winter. Nine months since he experienced pain under his heart, and strong palpitations, which were quickly followed by a diminution in the quantity of his urine, by œdema of the feet and ankles, and by urgent dyspnœa, which, as also the palpitations, were much increased by work, or any violent exercise. All these unpleasant symptoms were relieved by his club surgeon, Mr. Cartwright, and the œdema disappeared. Four months since they returned, and on applying to me he was again relieved by tonic and diuretic medicine, but I have no notes of his state at that time. For the last six months dyspnœa had greatly increased.

On admission into the Birmingham General Hospital he was seen to labour under general anasarca. His face was puffed and had a livid tint, particularly the lips; his eyes were rather injected. He complained of pain at the epigastrium, and of a very troublesome cough, which disturbed him at night, and was attended with copious expectoration of mucus. His pulse was ninety, small, feeble, and rather jerking. The urine was scanty, turbid, and contained much lithate of ammonia, but no albumen. The veins of the neck were very turgid, particularly on the right side, and exhibited broad undulations synchronous with the systole of the ventricles of the heart.

Percussion elicited a very clear sound all over the chest, the usual precordial dullness being imperceptible. The pulmonary sound was very faint, mixed here and there with bass-viol sounds, and small, fine muco-crepitant rattle. The impulse of the heart was moderate. A weak systolic rasp-sound was perceived, and was at its maximum around the left nipple, but was not heard at the top of the sternum. He died in three days.

Inspection. The veins of the neck and the lungs were gorged with blood, but there was no pulmonary apoplexy. There were patches of emphysema interspersed through the lungs, more particularly along the free margin, which overlapped the heart.

The pericardium adhered to every part of the heart by cellular tissue and clear gelatinous matter; it was, however, easily separated by the finger. The heart itself was not much larger than usual. There were patches of atheroma in the aorta. The corpora arantii of the aortic valves were thickened and enlarged, more particularly one of them, which projected considerably. The apices of the mitral valves were much thickened and corrugated, and one of them was only a quarter of an inch deep, and was divided into three or four portions, each with a separate cord, thus resembling a tricuspid valve. The tricuspid orifice nearly admitted all four fingers and the thumb, and measured six inches and a half in circumference. The valves were thickened and corrugated at their free edges, and attenuated at their bases. They were much shortened, the depth of one being less than an inch, of another half an inch, and that of the third still less.

The pericardium being so easily separated from the heart had probably not long become adherent. The state of the mitral valves could hardly have been produced otherwise than by original malformation, which would account for the habitual dyspnœa from birth. When dilatation came on, the mitral and tricuspid orifices would both admit of regurgitation, and hence the formidable train of symptoms. It is remarkable that the patient should, under these circumstances, have been twice so much relieved.

Case XLVI.

Bronchitis—Mitral and tricuspid regurgitation—Dropsy.

A gardener, æt. 74, with fine ruddy complexion, and of shortish stature, had enjoyed uninterrupted good health until twelve months ago, since which time he has taken cold upon every slight exposure to the weather. During the last three months he has suffered from dyspnœa, constriction of the chest, orthopnœa, and cough, at times suffocating, with scanty muco-purulent expectoration. Within ten days his legs began to swell, and on his admission into the Birmingham General Hospital they were much enlarged, and pitted deeply on pressure. His bowels were regular, and he passed a pint and a half of urine daily. His pulse was under 70. The radial artery was tortuous and felt like a cord. The chest sounded clear on percussion, except that the precordial dullness extended rather lower than usual. The pulmonary sound was accompanied by a mixture of bass-viol and cooing sounds, and mucous rattles. The impulse of the heart was feeble and slow. Both its sounds were prolonged and muffled, and were not heard beyond the precordial region.

In spite of treatment his symptoms became aggravated. The dyspnœa and orthopnœa increased greatly. His nights were restless. Anasarca gained the abdomen, and effusion into the left pleural cavity was detected by dullness on percussion varying with position, tubal breathing, and tremulous bleating resonance of voice. A gangrenous slough appeared on the right leg, and he sank in less than three weeks after his admission.

Inspection. The right lung adhered to the side; it was congested in its most dependent parts. The left lung was compressed to half its proper size by two quarts of serum in the left pleural cavity, and was congested. Portions of the lining membrane of the bronchial tubes were congested and softened. They contained much muco-purulent fluid.

There were about three or four ounces of amber-coloured serum in the pericardium. The heart was fatty, and nearly double its usual size from an increase of its left side. The fibre was firm and dense, but was the colour of brown paper. The cavity of the right ventricle was much diminished and encroached upon by the left. The tricuspid orifice measured five inches and a half in circumference. Its valves were short, and all connected together by reticulated tendinous fibres. The walls, carneæ columnæ, and septum of the left ventricle were all much hypertrophied. The posterior mitral valve was ossified, and the cords of both were short and thick. The circumference of the mitral orifice was four inches and a half.

The aortic valves had lost some of their elasticity, but were not opaque or thickened. The coronary arteries were ossified. The aorta, and indeed all the vessels of the arterial system, were lined with atheroma, and here and there by calcareous deposit.

Case XLVII.

Mitral obstruction and tricuspid regurgitation—Dropsy.

A bargeman, æt. 22, of athletic frame, who had been much exposed to wet and cold, and had been subject to rheumatism, was admitted into the Birmingham General Hospital labouring under great dyspnœa and orthopnœa, with harassing cough and mucous expectoration streaked with blood, pain at the heart, and palpitations. His face and lips were turgid and rather purple. He had an anxious expression of countenance and a hurried manner.

The precordial dullness was extensive. Shrill, cooing sound was heard all over the chest. The impulse of the heart was heaving, and violent palpitations were frequent. The systolic sound was prolonged, shrill, and raspish, and was most distinct below the nipple. The diastolic sound was short and clear, and was heard distinctly near the angle of the left scapula. Both sounds were heard in some degree over the whole of the chest.

Under the use of small doses of digitalis and nitre his health was much improved for a time, and the blood disappeared from his sputa; but in a short time all the bad symptoms returned, and, in addition, the veins of the neck became turgid and pulsated, and œdema of the legs appeared. The systolic murmur became stronger, the hemoptysis severe, and dyspnœa very distressing with orthopnœa; the urine more scanty, the palpitations most distressing, and he died five weeks after his admission.

Inspection. On opening the thorax, the lungs, which were very large, did not collapse; they were congested and very emphysematous, particularly at their free margins. In the upper and middle lobes of the right lung were three or four yellow, soft tubercles, rather larger than peas. The base of the left lung was of a yellowish red colour, uncrepitating, heavy, and friable. In one part of it was a patch of a grayish colour, from which bloody pus exuded on pressure. In another part of it was a patch of pulmonary apoplexy of the size of a pigeon's egg, with well-defined edges. The whole of this hepatized portion of lung was drier than in ordinary pneumonia.

The pericardium was thickened and opaque, but contained no serum. On the front of the heart were several thickish, milky patches; the organ was large, and both ventricles were dilated; the walls of the left ventricle being firm, red, and hypertrophied. The tricuspid orifice was six inches in circumference, and the valves of the ordinary size. The zone of the mitral foramen was thickened, and only admitted one finger to pass freely. The valves were thickly fringed with warty vegetations, which extended into the auricle over the left valve.

Case XLVIII.

Aortic and tricuspid regurgitation—Dropsy.

A labourer, æt. 33, of athletic frame and intemperate habits, accustomed to violent exercise, but who had never had rheumatism, was admitted into the Birmingham General Hospital. His legs were anasarcous, as also were the walls of the abdomen, but to a less extent. His face was tumid and bloated, his lips purple, his expression of countenance anxious. The respiration was very laboured, the shoulders being raised, and he could not lie down. He had pains at the region of the heart, and constant palpitations. His urine was scanty and loaded with lithates; the pulse soft and feeble. There was cough with aqueous expectoration. The external jugular veins were seen to undulate above the clavicles.

The lower half of the right side sounded dull on percussion, the level of the dullness varying with the position of the patient. The left side sounded dull from the fourth to the eighth rib, from the sternum to the outside of the nipple. The pulmonary sound over the upper two thirds of the right lung was coarse, and attended with cooing sound; over the lower third it was feeble; over the left lung it was accompanied by some muco-crepitant rattles. The impulse of the heart was felt over the dull space around the precordial region, and was quick, jerking, and tumultuous. The sounds of the heart were heard all over the chest; the systolic sound at a distance from the precordial region being dull, and the diastolic sound longer and less clear than usual. Below the left nipple the systolic sound was prolonged, and of an acute shrill rasp quality, extending up the course of the aorta. At the middle of the sternum near the left fourth cartilage, both sounds were raspish and prolonged.

His symptoms became aggravated, the dyspnœa greatly increased, and he died in seven days.

Inspection. The left lung was universally adherent to the chest, and had apparently been some time in this state. There were four pints of serum in the right pleural cavity. The lungs were congested. The lining membrane of the bronchial tubes was of a purple tinge. The pericardium was opaque and thickened, and contained five ounces of yellow serum. The heart was very large, and weighed twenty-six ounces; it was soft and flaccid, and had many milky patches on its surface. Its cavities were all greatly dilated, and contained much black blood. The walls were not much hypertrophied. Two of the aortic valves were thickened and shrivelled, but could fall back during the systole of the ventricle, but the third valve was transformed into cartilage, and rigidly stood out from the side of the vessel. The mitral valves were slightly thickened. The tricuspid orifice was full six inches in circumference, and its valves were of the ordinary size. The lining of the aorta up to its arch was thickened, and studded with atheroma and calcareous scales.

Case XLIX.

Mitral and tricuspid regurgitation—Emphysema—Dropsy.

A wire-drawer, æt. 56, had been a collier twenty years since. Nine years ago, after having taken repeated colds, he had an attack of rheumatism, and was laid up during nine months with swelling of the legs, thighs, and body, and has been asthmatic ever since. When received into the Birmingham General Hospital he had been ailing two months.

There was slight angular curvature of the spine. There was anasarca of the lower extremities and abdomen. His face was purple and bloated, and he laboured under great dyspnœa.

He had cough, with expectoration of thick muco-purulent fluid. The pulse was 84, firm and steady. There were strong venous pulsations on the left side of the neck. The precordial dullness to the left of the sternum was more extended than usual, but the sternum itself sounded very clear. The pulmonary sound was everywhere feeble, and was mixed with cooing sounds, and behind the left side with large mucous crackling. The sounds of the heart were clear and well defined. He was not relieved by any treatment; the dyspnœa and anasarca increased, and he died in thirteen days.

Inspection. The right lung adhered slightly, and the left lung extensively to the thorax. There was rather more than half a pint of clear, light-coloured serum in each pleural cavity, and a considerable quantity in the abdomen. The upper half of each lung was studded with emphysema; most of the dilated cells being of the size of a mustard seed, and one or two reaching the size of a horse-bean. The heart was firm and red, and covered with milky spots in the shape of strings of small beads. The auricles were both dilated, but the ventricles were of a natural size. One of the mitral valves was shrivelled up to a mere thick, ragged-edged fringe, not more than two lines deep, with a great many cordæ tendineæ much thickened, and three stiff, hardened, and much hypertrophied carneæ columnæ. The mitral orifice measured four and a quarter inches in circumference. Two of the tricuspid valves were in the same state as that of the mitral valve above described.

In all these cases the progress of the disease seems to have been from the left to the right side of the heart, the symptoms of venous obstruction coming on with the incompleteness of the tricuspid valves. In Case XLIX there was both mitral and tricuspid regurgitation unaccompanied by any murmur.

CASE L.

Diseased aorta, hypertrophy, and dilatation of heart—Tricuspid regurgitation—Pericarditis—Pleuritis.

A labourer, æt. 40, an habitual drunkard, having drank cold water when he was heated ten weeks since, began to feel palpitations at the heart, followed by pain across the epigastric region, dyspnœa, and cough with expectoration of thick yellow phlegm. For the last four weeks he has been unable to lie down, and has had œdema of the ankles seven or eight days, and scantiness of urine during three days.

Admitted into the Birmingham General Hospital, he said he had less palpitation, but had pain across the epigastrium, a bad cough with hardly any expectoration, distressing dyspnœa and orthopnœa; a little pain in the head and up the left side of the face. The legs were anasarcous, and the scrotum and abdomen slightly so. The urine was scanty and high coloured, not albuminous; the pulse was rather full and hard, under 100. There were violent pulsations of the arteries visible at the wrists. The respiration was 30, and its sounds were natural. There was extended dullness around the region of the heart, with heaving action of that organ; pulsation over each clavicle, and a thrill was felt on deep pressure over the sternum. The diastolic sound of the heart was very faint, and the systolic sound like that of the fourth string of a violoncello, its maximum being up the course of the aorta under the sternum. Digitalis was given and a blister applied. He got much worse, however, and in a few days the scrotum being greatly distended, was punctured by Mr. Freer. He was ordered to take half a grain of elaterium twice a day, with an ammonia mixture, which produced strong action of the bowels; and though he was distressed by vomiting, he was relieved, and the anasarca was greatly diminished. Eighteen days after this, venous undulations were plainly seen in the neck, chiefly on the right side. Percussion was good, except, as before, over the precordial region, and below the fifth ribs behind. The respiratory sound was loud in front, coarse behind; the heart's action feeble and slow; a fine rasp-sound was heard at the extreme left of the side between the seventh and eighth ribs; near the nipple to the left of the sternum a soft systolic bellows-sound was heard, which became raspish as the right of the sternum was approached, and musical up the sternum, its maximum being about the middle of that bone.

The dyspnœa increased, and his legs became red, and were attacked with gangrenous sloughs. He died six weeks after his admission into the hospital.

Inspection. The cellular tissue was loaded with serum, a few pints of which were found in the cavity of the abdomen. The lower third of the right side of the thorax was also filled with serum, in which flakes of yellow lymph floated. This space was bounded by pleura, covered thickly with reticulated yellow lymph; in one or two spots on the lungs it was an inch thick, and resembled concrete pus. This lung was carnified at its lower half, and engorged all over; there were a few patches of pulmonary apoplexy in it, and also in the lowest portion of the left lung, which was otherwise healthy and very voluminous.

The heart was very large, firm and red. About three ounces of serum were in the pericardium, and on each fold of this membrane were rough patches of yellow lymph, not adherent to each other; one patch, rougher and thicker than the others, was on the left side near the apex, and one on the corresponding part of the sac. It was not of very recent formation. The mitral valves were slightly thickened, but acted well, and were fully equal to close the mitral orifice, which was four inches in circumference. The aortic valves were prevented from folding back on the vessel from their free edges being in several places transformed into cartilage, but they acted so as to prevent regurgitation. The tricuspid foramen measured five inches round, and had only two valves, one of which was a deep and long one, and the other was less than a quarter of an inch in height. The aorta was slightly dilated, and was lined with atheroma and steatoma, in which were some calcareous concretions.

Case LI.

Mitral obstruction—Tricuspid regurgitation—Dropsy.

A lad, æt. 13, had a severe attack of rheumatic fever when five years old. Four weeks since he felt palpitation, and three days back he felt for the first time a pain under his left breast. When seen with Mr. Bracey, he could not lie down, and dyspnœa was distressing. The pulse was small and vibratory; there were strong palpitations under the xiphoid cartilage. From the second down to the seventh rib of the left side, the chest was bombed out an inch beyond the level of the other side. This space gave out a dull sound on percussion, and over it existed a purring thrill. A loud coarse bellows-sound was heard all over the chest.

In a month's time the projection of the left side had much decreased under the employment of iodine and mercurial frictions. A pulsation and thrill were then felt between the fifth and sixth ribs, alternating in time with another between the second and third ribs. In another month the veins of the neck became very turgid, the feet and legs became œdematous, and ascites followed. The urine was not coagulable by heat. Four months from my first visit he was extensively anasarcous; the whole of the left side of the chest sounded dull, and the respiratory sound could only be heard on that side close to the spine. He then died.

Inspection. The heart was enormous, extending up to the second rib, and more than two inches to the right of the sternum. The lining membrane of the left auricle was thickened and opaque, and was covered with patches of a horn-like substance, which extended into the mitral valves. These were much thickened, and would not allow the passage of two fingers; the bases of the aortic valves were also thickened; the right ventricle and auricle were immensely dilated and attenuated; the tricuspid foramen measured five inches and a half in circumference. One of the tricuspid valves had its cordæ tendineæ much thickened and shortened, so that it could not rise into the plane of closure; consequently, the valve was incomplete. The other organs were healthy.

Case LII.

Mitral obstruction—Tricuspid regurgitation—Dropsy.

A girl, æt. 14, had articular rheumatism, with some pain under the left breast, two years and a half ago. Six months after that she began to perceive palpitations, which have occasionally reappeared ever since, accompanied by depression, nausea, and dyspnœa on exertion.

When visited, with Mr. Jones, she complained of constant nausea, long and violent attacks of palpitation, occasional headaches, and a dry cough. The pulse was feeble and variable in size. The left side of the chest sounded dull from the second rib downwards, and over this space the respiratory sound was inaudible. A faint pulsation was felt between the fifth and sixth ribs, alternating with a similar one between the second and third ribs. A grating sound was heard with the systolic sound of the heart. Six months after this, turgescence and strong pulsations of the jugular veins were seen. The feet, legs, and abdomen became gradually and successively infiltrated. The urine became scanty, but was not coagulable by heat. A double grating sound was heard in the precordial region, and continued till her death, which took place nine months after she was first seen.

Inspection. There was much fluid in the thorax, pericardium, and abdomen. On either fold of the pericardium there were several rough patches, which felt like a calf's tongue. The heart was hypertrophied, and dilated to more than double the size of the fist. The cordæ tendineæ of one of the tricuspid valves were shortened and thickened. The tricuspid foramen measured nearly five and a half inches in circumference. The valves could not close it, a large space being left uncovered in the middle, not only from the inability of one of the valves to rise up to the plane of closure, but from the disproportion which existed between the size of the valves and that of the foramen. The lining membrane of the left auricle was thickened and opaque, and near the mitral valves was granulated. These valves were thickened, and adhered to each other at their bases, so that two fingers could not pass between them.

The other organs were healthy.

Case LIII.

Mitral and tricuspid regurgitation—Anasarca—Dropsy.

A lawyer's clerk, æt. 21, had from a child felt a fluttering at his heart, and dyspnœa, after being hurried in any way, and particularly after running. He had rheumatic fever at 14 years of age, ever since which time he has had severe palpitations. He had another similar attack of a slight nature at the age of 18, and since then he has gradually become more and more pigeon-breasted.

When first seen he was labouring under great dyspnœa, The precordial space was dull over a larger extent than usual, and it was bombed out. There was no tumefaction or pulsation of the veins of the neck. The pulse was small. The systolic sound of the heart was accompanied by a very shrill musical saw-sound, the maximum of which was under the sternum, a little above the level of the nipple. It was continued, as it were, into the diastolic sound, so that the systolic sound terminated with a kind of jerking increase.

He was occasionally seen and relieved from time to time for several months; chiefly by belladonna frictions, ether, expectorants, &c. At length he complained of great constriction across the epigastric region, and of his feet being constantly cold.

When seen in consultation with Dr. Skerrett, the pulse was a little quick and sharp, and the precordial dullness extended. A musical saw-sound was heard during the systole of the heart, and was at its maximum under the left nipple, its intensity diminishing upwards from this point. It was heard plainly at the lower angle of the left scapula, but with less shrillness than in front of the chest. No diastolic sound could be heard. A strong purring thrill was felt around the left nipple, and towards the outer side of the chest. Soon after this the veins of the neck became turgid and pulsated, œdema of the feet and ankles appeared, and was quickly followed by general anasarca and death.

Inspection. The cellular tissue was loaded with serum. The lungs were engorged. The heart was dilated, particularly both the auricles. The mitral valves were thickened and shrivelled, and their free edges fringed with long horny vegetations, one or two nearly an inch long, so that they could not close the mitral orifice, which was four inches and three quarters in circumference. On one of the tricuspid valves was a bundle of soft, red vegetations, as large as a bean. The orifice was five inches in circumference, and the valves rather small.

In Case L there was visible pulsation of the arteries, without aortic regurgitation, which depended, perhaps, on hypertrophy. The musical termination of the systolic sound might have been caused by the vibration of the cartilaginous valve, which could not lie flat. The fine rasp-sound heard at the outer part of the side was produced by friction of the opposing surfaces of the pericardium, thickened by lymph. It is indeed often difficult to distinguish between valvular murmur and this friction-sound. In Case LII it had a grating character.

In all these cases symptoms of pulmonary obstruction first appeared, and were then followed by those of general obstruction, which was ushered in by venous engorgement of the neck.

Case LIV.

Aortic obstruction—Tricuspid regurgitation—Slight anasarca.

A gun-stocker, æt. 60, a patient of the Birmingham Dispensary, felt palpitations of the heart when in bed a year ago, which occasionally reappeared for six or seven months, and then ceased; great dyspnœa then came on. He had lost flesh for three weeks, and for three days had pain below the left breast. The veins of the neck pulsated most violently, and felt as firm and hard as arteries. The pulse was converted into a tremulous vibration. There was cough, with expectoration of much clear mucus, sometimes streaked with blood. The chest sounded dull about three inches around the heart. One coarse, hollow, and amazingly prolonged sound was heard synchronous with the ventricular systole, and its maximum was to the right of the lower part of the sternum. Cooing sounds were heard over the chest. There was slight œdema of the ankles and of the legs. Five weeks after this he was carried off by an attack of acute bronchitis.

Inspection. The body was emaciated, and presented no traces of anasarca. The veins of the neck were very much distended with dark blood. The pericardium was opaque and thickened, and had on its cardiac portion a patch of soft yellow lymph as large as a crown-piece. The heart was very firm, and was nearly three times the size of the fist, from general hypertrophy and dilatation. The aortic valves were unfolded, grown together, and calcareous, so that the passage for the blood was contracted into a small triangular opening, which could barely admit a quill. The aorta was studded with calcareous scales. The arch was slightly dilated, and its middle coat easily torn. One of the tricuspid valves adhered to the right ventricle. The tricuspid foramen measured five inches in circumference. The other organs were healthy.

Case LV.

Aortic obstruction—Tricuspid regurgitation.

An iron-caster, æt. 61, had oppression of the chest and indigestion for twelve months. He complained of troublesome cough and distressing dyspnœa. The pulse was very small and thread-like. An obscure thrill was felt to the right of the upper part of the sternum. A single prolonged rasp-sound was heard, its maximum being over the same spot where the thrill was felt.

Four months after this the pulse in each wrist was reduced to a mere flutter; and in nine months more the intensity of the rasp-sound was much diminished. The whole of the right side of the chest sounded dull, and the respiratory murmur of that side could only be heard up the spine. He could not lie on the left side. There was extensive dullness around the heart.

In six months the veins of the neck were almost varicose; the ankles became œdematous, and in another month general anasarca appeared. The urine was not coagulable by heat. A little before his death, which took place nearly two years after my first visit, a slight fluctuation could be occasionally detected in the jugular veins.

Inspection. The right side of the chest contained a gallon, the left a pint of fluid; and there was a large quantity in the abdomen. The size of the heart was truly astonishing; it projected for some distance under the sternum into the right side of the chest. All the cavities were uncommonly dilated, and the walls of the right ventricle were attenuated. There were only two aortic valves which were hard and calcareous, and stretched tightly across the orifice of the vessel. The free edge of the smaller valve was cartilaginous, so that it could slightly recede, and allow a slit for the passage of the blood from the heart. These valves had long calcareous bases which extended some distance into the ventricle. The tricuspid foramen measured six inches in circumference; its valves were small, and totally incompetent to close it. There was atheroma in all the great arteries.

The other organs were healthy.

The nature of the disease of the aortic orifice in both these cases proves that it must have been of long standing, and yet but little inconvenience had been felt until within a short period of death.

It is unfortunate that the size of the mitral orifice should not have been noted and accurately compared with that of the valves; for it is hardly possible to conceive that any valves could have effectually closed orifices so much dilated as these were; and yet they appeared to do so on viewing them. As long as the disease was confined to the left side, as in Case LV, there was no œdema, but when the tricuspid orifice began to dilate, as proved by the turgescence of the external jugular veins, it soon appeared. In Case LIV the progress cannot be thus traced, as these veins were dilated when the patient was first seen. It is very remarkable, that with this extreme venous distension so little anasarca should have been present, so that after death the traces of it had disappeared from the body. It was on this account difficult to refrain from placing this case in Class I.

The adhesion of one of the tricuspid valves must have been of inflammatory origin. Other similar cases will be given, in which no other traces of inflammation beyond the adhesion could be detected. This case tends to prove that strong regurgitation may occasionally take place through the tricuspid orifice, without for a time giving rise to any considerable amount of anasarca.

In both cases a very lengthened systolic rasp-sound was heard, which proved that the contraction of the ventricle was prolonged. By this means the blood was squeezed through the contracted aortic orifice, and thus the circulation was kept up, and the stagnation of the blood in the heart and lungs to a certain extent prevented. It will be remembered that a similar sound was heard in Case XLII, and was produced by the passage of blood through the narrow mitral orifice.

CASE LVI.

Adherent pericardium—Mitral obstruction—Dropsy.

A girl, æt. 8, was for nine months afflicted with articular rheumatism in the elbows, wrists, and finger-joints, the latter being quite distorted with swellings, some of which were as large as nuts. These gradually disappeared, and after some months' time she was in tolerably good health. Four months after this she took cold, from sitting in a cold damp place for some hours. She became an out-patient of Addenbroke's Hospital, Cambridge, but after a week was too ill to attend, and I visited her at her own home. She was suffering from intense dyspnœa and orthopnœa, and complained of great pain and tightness across the region of the heart. There was slight œdema of the ankles. The action of the heart was violent, and its pulsation strongly marked under the left nipple. About this point a slight systolic bellows-sound was heard. The chest sounded well on percussion, and the respiratory sound was natural. Leeches were applied to the precordial region, and this was followed by strong mercurial frictions. Effusion, however, gained the abdomen, and the patient quickly sank.

Inspection. The pericardium was universally adherent to the heart. The left auricle was greatly dilated, and filled with a black clot. The mitral valves were fringed with osseo-cartilaginous vegetations, with sharp-pointed spiculæ, oné of which was a quarter of an inch long.

This case occurred in 1834, when shortening of the cordæ tendineæ would certainly have been overlooked by me. If, however, the valves were perfect, they must have closed the tricuspid orifice, and then this would be a case in which dropsy occurred without any apparent obstruction to the circulation on the right side of the heart. I have met with two other such cases in the practice of others.

In the cases which follow, there was no trace of disease on the left side of the heart.

CASE LVII.

Tricuspid regurgitation—Pulmonary apoplexy—Dropsy.

A married lady, æt. 29, had not enjoyed good health since an attack of rheumatic fever in early youth. She had for four months suffered from dyspnœa, during which time she constantly expectorated clear mucus, sometimes mixed with dark blood, and had occasional attacks of severe pain in both sides, with fever.

When visited in consultation with Mr. S. Partridge, she could not lie flat; her urine was not coagulable by heat. In about a month after this her legs began to swell, and a slight systolic bellows-sound was heard over the precordial region, when she was hurried or excited. The veins of the neck, which had been always rather full, now became very turgid, and pulsated strongly. The œdema of the legs increased, ascites appeared, and she died, the subject of extensive anasarca, three months after my first visit.

Inspection. There was a considerable amount of serum in both sides of the chest, and in the abdomen. Some patches of circumscribed pulmonary apoplexy were seen in the lungs.

The heart was more than twice the size of the fist, being hypertrophied and dilated throughout. Two flaps of the tricuspid valves acted imperfectly, one being partially and the other entirely adherent to the right ventricle. The tricuspid foramen was greatly dilated, and its circumference measured five inches and a half. An immense transparent coagulum, of the consistence of jelly, was lying in it, and extended into both the auricle and the ventricle. The other organs were healthy.

CASE LVIII.

Tricuspid regurgitation—Dropsy.

A girl, æt. 14, pigeon-breasted, had rheumatic fever twelve months since, with very severe pain under the left breast, and distressing dyspnœa.

When visited with Mr. Jones she complained of great dyspnœa, and occasional severe pain in the precordial region.

The urine was not coagulable by heat. On the left side the chest sounded dull from the second rib downwards. A purring thrill was felt under the left nipple. At the same spot a bellows-sound was constantly heard, and for some distance around it. In two months' time the veins of the neck were observed to fluctuate obscurely. In another fortnight the ankles and legs became œdematous, ascites soon followed, and in three months from my first visit she died extensively anasarcous.

Inspection. A large quantity of clear lemon-coloured fluid was found in the abdomen, about a pint in each pleural sac, and three ounces in the pericardium. The lower part of each lung was slightly carnified, they were otherwise healthy.

The heart was dilated to nearly double its natural size, the walls retaining their usual thickness. The valves were not thickened, but a part of the tricuspid valve was glued down to the side of the right ventricle, and thereby prevented the closure of the foramen, the circumference of which measured four inches and a quarter. The other organs were healthy.

Case LIX.

Tricuspid regurgitation—Dropsy.

A single woman, æt. 57, a cook, had enjoyed good health up to the age of 34, when menstruation ceased, and she then suffered from dyspnœa and profuse leucorrhœa. Eighteen months since she first felt a dull pain in the right hypochondrium, which was attended with considerable derangement of the stomach, and whitish-coloured stools. She was at that time consuming a great quantity of animal food, ale, and spirits. Her symptoms were relieved by treatment. A few weeks since her feet and ankles, and subsequently her abdomen swelled, and she was received into the Birmingham General Hospital.

There was little or no dyspnœa, and no cough. The chest sounded clear on percussion, except that the dullness in the precordial region was more extended than usual. The respiratory sound was natural. The heart's action was weak and irregular, and its sounds rather obscure. There was great turgescence, and some pulsation in the veins of the neck. The urine was not albuminous.

With rest, and under the use of tonic and diuretic medicines, and a tolerably generous diet, the anasarca disappeared, and she left the hospital and took a cook's place.

Six months after this she was again admitted. Her legs and abdomen were greatly distended with fluid. The stools were pale and offensive. The urine yellowish and turbid, abounded with phosphates, but contained no albumen. The veins of the neck were greatly distended and pulsated. She could only sleep in a semi-reclining posture, and laboured under great dyspnœa. The dullness over the precordial region was very extensive, and there was also dullness at the bottom of each side of the chest varying with position. There was no cough or expectoration. The pulmonary sound was clear but rather coarse, except at the bottom of each lung, where it was very feeble, and occasionally mixed with a fine muco-crepitant rattle. The sounds of the heart were both distant and muffled, running into each other from the great irregularity of the heart's action. This time medicines made no impression on her; her symptoms continued to become more and more aggravated, and she died in less than a month.

Inspection. The abdomen, and the integuments of the body generally, were greatly distended with fluid, and a considerable quantity existed in both pleural cavities. The lungs were healthy.

There were four ounces of fluid in the pericardium. The heart was large, the right auricle being more particularly dilated. There was a great deal of fat in the walls. The sigmoid valves acted well, although the corpora arantii of two of them were a little thickened. The tricuspid orifice measured four inches in circumference. The back valve, as well as a thick fleshy column fixed to its apex without any cord, were closely glued to the ventricle. The edges of the other two were thickened and shrivelled; the height of one was half an inch, and the other a quarter of an inch. There were milky patches on the lining membrane of the right ventricle.

The liver was rather hard. The kidneys were lobulated, and their appearance was slightly granular on being laid open.

Case LX.

Tricuspid regurgitation—Dropsy.

A nurse, æt. 48, married, had been asthmatical for eight or nine years; but for the last four years she had suffered greatly in winter, and in foggy weather she felt "bloated up for breath." About six months since her legs began to swell, and she had pain at the heart and urgent dyspnœa. On being admitted into the Birmingham General Hospital, her symptoms chiefly consisted of broad undulations in the veins of the right side of the neck, in addition to the pain mentioned and general anasarca. She was relieved by diuretic and tonic medicines, and by belladonna frictions over the heart, and left the hospital, every trace of venous pulsation and anasarca having disappeared.

Some time after this she applied again as an out-patient. She had been exerting herself, had been exposed to alternate heats and chills, and anasarca had reappeared; after any unusual exertion, her hands felt benumbed until they had been rubbed. Upon stooping and rising again, she felt a pain under the xiphoid cartilage. Her appetite was bad. Her breathing was but very little oppressed. The pulse was small but regular. She had a troublesome cough, with some wheezing and expectoration of thick blackish gray mucus. The action of the heart was natural. Strong broad venous pulsations were observed in the neck, stronger on the right than on the left side. The pulmonary sound was coarse, accompanied in most parts of the chest by bass-viol and cooing sounds. On the left side there was some moist crackling, the pulmonary sound commencing with crackling, and terminating in cooing and bass-viol sounds. The diastolic sound of the heart was stronger and coarser than usual, and was heard all over the chest; but there was no other abnormal cardiac sound up to the time of her death, which took place in six weeks' time, the anasarca gradually increasing.

Inspection. There was much serum in each pleural cavity, and a very little in the pericardium. The lining membrane of the air-tubes was thickened in places, as in chronic bronchitis.

The right auricle of the heart was dilated. The tricuspid orifice measured five inches in circumference, and all the valves had very short thick cordæ tendineæ, so that the valves could not rise up into the plane of closure. The aortic valves were thickened, but acted well.

The adhesion of one of the tricuspid valves to the ventricle which occurred in these cases, as well as in Case LV, must have allowed regurgitation, which was doubtless much increased by the dilatation of the right side; in Case LVII arising from congestion and pulmonary apoplexy of the lung; in Case LVIII from inflammation accompanying rheumatic fever; and in Case LIX, from that subacute form of inflammation to which spirit-drinkers are subject, and the traces of which were here found in several organs. The inflammatory origin of the adhesion was clearly marked in this case by the thickened milky patches on the lining membrane of the ventricle near the adherent valve.

The dyspnœa, which in all these cases preceded the dropsy, arose, in Cases LVII and LX, from the state of the lungs, and in the other two cases possibly from the effect of inflammation on the muscular walls of the heart.

The influence of rest, diet, and treatment in removing the dropsy for a time, in Case LIX, while its cause remained untouched, is remarkable.

CASE LXI.

Diseased arteries—Tricuspid regurgitation—Dropsy.

A boot-maker, æt. 46, an old soldier, who had lost the right leg above the knee, had been accustomed to drink spirits to excess. Nine months since he felt a pain under the sternum, followed by dyspnœa, both of which have continued ever since. When visited with Mr. Clark the dyspnœa was most distressing: he was unable to lie down, and had a strong sensation of constriction across his chest. There was œdema of the leg and thighs, and some ascites. The jugular veins pulsated strongly. There was dullness on percussion for some inches around the precordial region. The respiratory sound was natural. The action of the heart was heaving, and its sounds distant. The pulse was very full and hard, and its stroke remarkably quick and sharp, which character it retained till death took place, three weeks after my first visit, having been preceded by the expectoration of dark clotted blood for two days.

Inspection. There was serous effusion in the chest and abdomen. A patch of pulmonary apoplexy, as large as an orange, existed about the middle of the concave portion of the right lung, near the pericardium, and was continued gradually into the healthy portion of the lung.

The heart was enormously hypertrophied and dilated. On its surface was a patch of thick, yellow lymph. The dilatation of the right cavities was very great: the circumference of the tricuspid foramen was five inches and three quarters; one of the valves had short, thickened cordæ tendineæ, they could not close the foramen. The aortic valves were thickened, but acted well. There were many patches of atheroma under the lining membrane of the left auricle. The aorta was a mass of disease, being thickened and puckered, and in many places denuded of its lining membrane, and all the large arteries were similarly diseased.

The liver was of a nutmeg colour. The kidneys and other organs were healthy.

Case LXII.

Aortic obstruction—Tricuspid regurgitation—Dropsy.

A wood-turner, æt. 62, had an attack of acute bronchitis eighteen months ago, since which time he has felt a tightness of the chest; for the last nine months dyspnœa, and lately palpitation. Six weeks ago his legs swelled for a few days; this went off, and returned three weeks since. Dyspnœa was most urgent. The jugular veins were seen to pulsate chiefly on the right side, but were not turgid. The urine was not coagulable by heat.

There was dullness, more extensive than usual, around the region of the heart. The pulmonary sound was coarse in places, with slight cooing. The action of the heart was tumultuous, heaving, and irregular. The pulse was irregular, but moderate. The sounds of the heart were feeble and obscure, but on the right side of the sternum the systolic sound was louder, and slightly coarse and prolonged. In two or three days' time he began to expectorate dark, clotted blood. This, with increasing dyspnœa and anasarca, continued up to his death, which took place three weeks after my first visit.

Inspection. The legs and thighs were anasarcous; a little fluid was found in the abdomen. At the inner edge of the back, and lower portion of the upper lobe of the right lung (both of which were otherwise healthy), was a mass of pulmonary apoplexy, of the size of an orange, with abrupt edges. The heart was immense, and measured fifteen inches around the outside of the base of the ventricles. It was generally hypertrophied, and dilated, firm, and red. Within the folds of one of the aortic valves was a patch of semi-osseous matter, which did not prevent the free action of the valves. The circumference of the tricuspid foramen measured full six inches. One valve was large, the other two small. They were roughened by small hard substances, rather less than split mustard seeds, particularly on their free edges. The coronary vessels were large, but healthy, and the aorta was studded with numerous soft elevations produced by atheromatous deposit. The other organs of the body were healthy.

Case LXIII.

Endocarditis—Tricuspid regurgitation—Dropsy.

A porter, æt. 32, was attacked with acute rheumatism twelve years ago, and again eight years since, when he had very severe pains in the left breast, troublesome cough, and dyspnœa. Eighteen weeks previous to my visiting him his ankles began to swell, then his legs and abdomen. There was much dyspnœa. The urine was not coagulable by heat. The pulse was hard, sharp, and vibratory. The jugular veins were turgid, and pulsated.

There was dullness on percussion at the bottom of each side of the chest. The systolic sound of the heart was accompanied by marked bellows-sound. In a few days' time intense pain at the precordial region suddenly made its appearance, with most distressing dyspnœa. The pulse rose to 120, and was very sharp, hard, and full. Venesection having been freely employed, and having been joined with the administration of digitalis and mercurial frictions, the acute symptoms were removed; the anasarca and the pulsation of the jugular veins disappeared. The pulse remained very hard and vibratory.

In a few weeks venous pulsations of the neck, rapidly followed by anasarca, reappeared, and he gradually sank.

Inspection. Much fluid was found in the abdomen and thorax, and four ounces in the pericardium. This membrane was thickened and opaque in patches.

The heart was generally hypertrophied, and dilated to more than twice its natural size. The tricuspid valves were a little thicker and larger than usual, but could not nearly close their foramen, which would admit four fingers and the thumb nearly up to the knuckles, and which measured nearly seven inches in circumference. The mitral valves were thickened, but acted well. The lining membrane of the left auricle was thickened and opaque, and was studded with several horn-like patches. On one of the aortic valves was a patch of soft, reddish, recently-organized false membrane, of the size of a fourpenny-piece. The lining membrane of the aorta seemed swollen, and puckered unevenly. The other organs were healthy.

CASE LXIV.

Tricuspid regurgitation—Emphysema—Dropsy.

A labourer, æt. 55, had, during several winters, cough, with wheezing, dyspnœa, and expectoration of pearly mucus; and last winter he had œdema of the legs. In the spring all these symptoms, except the expectoration, disappeared.

When visited at the commencement of another winter, he was found to have been ill for some weeks. There was great œdema of the legs, thighs, and scrotum. His urine was scanty and high coloured, and not coagulable by heat. The respiration was hurried, and he had frequent palpitations. There was dullness on percussion around the heart. Muco-crepitant rattle was heard all over the chest. The sounds of the heart were sharp and clear, and were heard over the whole thorax, but seemed distant when listened to over the precordial region. He died in fourteen days.

Inspection. The lungs were gorged with serum. Large patches of emphysema existed at the summit of each lung; some cells were of the size of a horse-bean, and were bounded by tough, inelastic walls.

The heart was dilated to three times the size of the fist, its parietes being of the usual thickness. The right ventricle and auricle were particularly large, and the tricuspid foramen measured six inches in circumference. Its valves were small and thin, and could not close the aperture.

The other organs were in a healthy state, except that the lining membrane of the stomach was of an uniform bright scarlet colour, and rather mammillated at its great curvature.

There was no structural change in the valves of such a nature as to interfere with their action in any of these cases; but in every one of them there was dilatation of all the cavities of the heart and of the tricuspid orifice; and in the first three there was also hypertrophy. In these three cases the dilatation probably arose from inflammation; in Case LXIV, as in Case XXXIX, from pulmonary engorgement. Tricuspid regurgitation was, therefore, the only cause of the obstruction of the general circulation which was found in these cases.

CASE LXV.

Softened heart—Tricuspid regurgitation—Dropsy—Death from apoplexy.

A woman, æt. 60, some years ago had an attack of acute rheumatism, since which time she has had a cough with copious clear expectoration. During the last seven months she has suffered much from dyspnœa and palpitations.

When seen, with Mr. Clark, her pulse was feeble and irregular, the veins of the neck were much swollen, presented a knotted appearance, and pulsated faintly. There was dullness for a large space around the precordial region. The sounds of respiration and of the voice were natural. Bellows-murmur was heard over the precordial region. The heart's action was irregular; the urine was not coagulable by heat. The ankles were œdematous, and serous infiltration was becoming apparent in the thighs, when she was suddenly seized with apoplexy, and died in twelve hours.

Inspection. A great quantity of black coagulated blood was spread over the surface of each hemisphere of the brain, between the dura mater and the arachnoid membrane. There was a clot with laceration of the interior of each hemisphere.

The heart was dilated to an enormous size; the walls, of the usual thickness, were soft and flabby, of a yellow, mottled colour, and crepitated under the fingers like a piece of lung. The valves were not diseased. The circumference of the tricuspid foramen measured nearly six inches, and its valves could not effect its closure. The other organs were healthy.

Case LXVI.

Congestive apoplexy—Diseased arteries—Tricuspid regurgitation.

A coach-harness filer and dresser, of temperate habits, was admitted into the Birmingham General Hospital. His wife stated that five months before his admission he was seized, while at work, with what he described as a weakness in his right arm. This was speedily followed by a fit, in which he struggled violently for half an hour, after which he became delirious, and talked and raved incessantly for three or four days, during which time he could not answer any questions. He was much weakened by this attack, and in ten days' time he complained that his right side and limbs were weak and cold. He partially recovered from this state in the course of a month, but had constant headaches, which did not prevent him from resuming his employment.

A month since he had another struggling fit, succeeded by delirium, which lasted for four days. On recovering from this state, his articulation was slightly affected, his hearing impaired, and his sight rather dimmed, and he had less power in the right than in the left half of his body.

The notes of his state on admission, &c., are mislaid. He died from an attack of cerebral apoplexy.

Inspection. All the vessels of the membranes and substance of the brain were very full of blood, and those at the base of it were ossified. The arachnoid membrane over the hemispheres was opaque, and the white substance of the brain was of a dusky colour. The upper layers of the floor of each ventricle in the middle were rather softened. An ounce or two of serum escaped from the base of the cranium and spinal cord.

The lungs were pale and bloodless.

The heart was large, in consequence of the dilatation of its right side, while the cavity of the left ventricle, the walls of which were red and firm, and of the natural thickness, could not contain more than an almond. The circumference of the tricuspid foramen measured five inches. The aortic valves were thick and cartilaginous at their bases, but acted well. The mitral orifice admitted two fingers. The aorta was much diseased, being thickened and roughened with atheroma and calcareous scales, as also were the arteries of the neck and arms. In one spot of the aorta the inner coat was totally, and the middle coat was partially, destroyed by ulceration.

Case LXVII.

Tricuspid regurgitation—Congestive apoplexy.

A railway sub-contractor, æt. 34, had been under my care with symptoms of hypertrophy of the heart, which followed rheumatic fever. There were no signs of any valvular disease. Having been one day engaged during some hours in superintending the laying of rails, and having very often stooped down to run his eye along the line, he was suddenly taken ill, and expired in five minutes.

Inspection. The heart was red, large, and firm, being hypertrophied and dilated. The tricuspid orifice measured five inches in circumference.

The veins of the brain were gorged with dark blood, an immense quantity of which issued from the jugular veins.

Tricuspid regurgitation must also be considered to have been the cause both of the dropsy and apoplexy which occurred in these cases. Had this latter affection depended on a diseased state of the arteries in Cases LXVI and LXVIII, death would probably have been produced by their rupture; and in Case LXV they were not diseased.

The following Table exhibits the manner in which the various lesions of the heart were combined with each other in the above cases.

TABLE I.

THIRTY-SEVEN CASES OF DISEASED HEART.

11 *Cases.* Class I.—*Heart disease, without obstruction of the general circulation.*

CASE.	Adhesion of pericardium.	AORTIC.		MITRAL.			PULM. ART.		TRICUSPID.			DILATATION.		HYPERTROPHY.	
		Regurgitation.	Obstruction.	Regurgitation from disease.	From dilatation.	Obstruction.	Regurgitation.	Obstruction.	Regurgitation from disease.	From dilatation.	Obstruction.	Left.	Right.	Left.	Right.
XXXI		*		*											
XXXII				*	*							*			
XXXIII	*			*		*	*					*			
XXXIV						*							*		
XXXV	*					*									
XXXVI			*			*						*		*	
XXXVII			*									*	*	*	*
XXXVIII												*	*	*	*
XXXIX												*	*	*	*
XL				*		*						*		*	*
XLI				*		*						*		*	

26 *Cases.* Class II.—*Heart disease, with obstruction of the general circulation.*

XLII		*		*		*			*	*		*	*	*	
XLIII	*					*				*			*		
XLIV	*					*			*	*		*	*		
XLV	*		*	*					*	*					
XLVI				*	*				*	*		*			
XLVII						*				*		*	*	*	
XLVIII		*	*							*		*	*	*	*
XLIX				*	*				*						
L			*						*	*		*	*	*	*
LI						*			*	*		*	*		
LII						*			*	*		*	*	*	*
LIII				*	*	*				*	*	*	*		
LIV		*							*	*		*	*	*	*
LV		*								*		*	*		
LVI	*					*						*			
LVII									*	*		*	*	*	*
LVIII									*	*		*	*		
LIX									*	*			*		
LX									*	*			*		
LXI										*		*	*	*	*
LXII										*	*	*	*	*	*
LXIII										*		*	*	*	*
LXIV										*		*	*		
LXV										*		*	*		
LXVI										*			*		
LXVII										*		*	*	*	

The annexed Table exhibits the lesions of the heart which were revealed in the inspections above detailed, and also in those of the bodies of 118 other persons who died in the Birmingham General Hospital.

TABLE II.

ONE HUNDRED AND FIFTY-FIVE CASES OF DISEASED HEART.

	CLASS I. 39 Cases. Without obstruction of the general circulation.	CLASS II. 116 Cases. With obstruction of the general circulation.
Incomplete aortic orifice . .	9	16
mitral ditto . .	16	37
aortic and mitral do.	8	6
pulmonary orifice .	*4	1
Tricuspid obstruction . . .	1	0
Tricuspid regurgitation from	5	106
disease 1		5
dilatation 0		60
both *4		41
Dilation of left ventricle . .	10	9
right ditto . . .	†3	22
both	†15	83
Hypertrophy of left ventricle .	7	13
right ditto . .	1	2
both	12	32
Adherent pericardium . . .	6	13
	Of the cases in this class death resulted from pulmonary obstruction in . . 20 Pulmonary disease in 3 Both in . . . . . 9 Sudden stoppage of the heart in . . 10	Of the cases in this class there was anasarca in . . . 102 Ditto and cerebral apoplexy in . . 7 Cerebral apoplexy alone in . . . 7

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* In three of these cases there was open foramen ovale.

† In fifteen of these cases there was also dilatation of the tricuspid orifice, but in twelve of them the valves were so large that they closed the orifice and prevented regurgitation, and in the other three the foramen ovale was open.

An examination of the foregoing cases shows that the principal alterations of the muscular walls of the heart which affected its contractile power, were *Hypertrophy*, *Attenuation*, and *Softening*.

When the natural colour was retained in the hypertrophied walls of the ventricle, I was unable to detect any difference in the minute structure of the muscular fibre from that of the walls of a perfectly healthy heart. Measured by the aid of Powell's micrometer eye-piece, under a magnifying power of 500 diameters, the average breadth of the fasciculi was about 1-400th of an inch in both cases. Vogel,[1] either from his own researches or those of Henle, has arrived at the same conclusion; and states that "the volume of muscle is increased, without the single fasciculi gaining in thickness; hence it must follow that their number is increased, and that new ones have arisen amongst those previously existing."

The walls of the ventricles have not been described as hypertrophied, except by extent, unless the thickness of the left ventricle, about midway between the base and the apex, exceeded six lines in the male, and five lines in the female; nor unless that of the right ventricle at the same spot measured above three, or two and a half lines, according to sex; these measurements being the result of Bizot's researches.

Attenuation of the muscular walls of the ventricles was, in almost every case, the result of dilatation of these cavities, produced by various causes. The most common alteration, however, consisted in the union of hypertrophy with dilatation.

A softened or flabby state of the ventricles was generally connected with an alteration of their appearance. Thus in some cases a great part of the muscular fibre, particularly on the right side of the heart, was covered, and sometimes replaced by masses of fat. In others the walls, either in patches or throughout their whole extent, presented a yellow, brownish, mottled appearance. Viewed under the microscope, this was found to arise from a deposition of fat-globules, often very small, surrounding and interspersed with the proper muscular fibre.

Another very common appearance was that which I have been in the habit of denominating the brown paper heart, from its walls assuming the colour of that substance. This was almost always associated with hypertrophy. It is possibly this state of the organ which is referred to by Rokitansky, and which he states to arise from the presence of minute fat-granules interspersed among the primitive fibres of the heart's muscles. I have ascertained that the muscular fibres are affected in these cases, being often more like the involuntary muscles in appearance, from having lost their transverse striæ. I have sometimes, also, seen a portion of the substance in the field of the microscope disappear on the addition of ether, but not in all cases. The examination of muscles in this state should be repeated by more experienced investigators. It will probably then be found that this appearance does not always depend on the presence of fat-granules. It may possibly be produced in some cases by inflammation, and in others by constitutional derangements, similar to those which induce atheromatous deposits, especially as such deposits were frequently found in the coronary arteries in these cases.

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[1] Op. cit. p. 184.

When softening is joined with attenuation, it tends of course still further to weaken the contractile power of the heart's muscles; but when combined with hypertrophy it acts antagonistically to it, and therefore in computing the effects of hypertrophy, the state of the walls in this respect must be taken into account.

A perusal of these cases further shows, that the chief alteration of the orifices and valves of the heart were of such a nature as to prevent the proper action of the valves, to diminish the size of the orifices, or else so to widen them that the valves could not close them.

At the mouths of the aorta and pulmonary artery the valves were shortened and thickened, adherent to each other, and to the sides of these vessels, or the orifices were contracted by calcareous deposits. At the auriculo-ventricular foramina similar alterations existed, besides which, the chordæ tendineæ were often so shortened, that the valves could not rise up into their plane of closure; and sometimes these orifices were dilated to such an extent, that the valves, although in a healthy state, could not close them.

The effect of some of these alterations was to oppose obstacles to the onward course of the blood, and of others to allow its regurgitation. Their causes were threefold:—

1. Atheromatous and calcareous deposits. 2. Inflammatory thickening, deposition of horny and pseudo-cartilaginous patches, vegetations, and adhesions. 3. Simple dilatation, almost always involving both ventricle and auricle; and induced either by inflammatory action, various debilitating causes, softening of the heart's muscles, or mechanical obstacles to the onward course of the blood.

Atheromatous and calcareous deposits were almost entirely confined to the left side of the heart, except when the foramen ovale was open. Simple dilatation of an orifice was more frequent, and carried to a greater extent on the right than on the left side. The results of inflammation were seen equally on both sides.

It will now be necessary to trace the effects of these morbid changes on the health and life.

Alterations of the walls of the heart. The effect of *hypertrophy* on the health may be favorable or unfavorable, according to the circumstances under which it occurs, and the other lesions with which it is complicated. Thus the natural hypertrophy which has been shown by Bizot to increase with age, manifestly favours the circulation, which is often much affected by the inelastic state of the vessels, and other circumstances attendant on the progress of age. So also when it occurs in connexion with obstruction or regurgitation at the aortic orifice, it effects a sort of compensation by forcing the blood into the aorta with increased momentum. And the same takes place when there is obstructive disease at the auriculo-ventricular valves; for the force of the diastole of the ventricles is in proportion to that of the systole, and the stronger it is, the greater is the tendency towards the production of a vacuum, and therefore the greater is the force with which the blood rushes in through the obstructed auriculo-ventricular orifice.

Hypertrophy may act unfavorably on the health, by retarding the venous circulation when regurgitation takes place through the auriculo-ventricular valves, or by producing arterial congestion of the lungs, or of the various viscera which receive their supply of blood from the general circulation. The former of these effects was probably very great in Cases XXXIII and others, when the blood was forcibly driven back through the mitral orifice on the pulmonary veins; and in Cases L, LII, LIV, and several others, in which the force of tricuspid regurgitation was increased by hypertrophy of the right ventricle.

Arterial congestion, however, does not appear to have occurred in these cases in the same proportion as has been observed by some other writers, or at least not to have produced serious consequences in so many instances. Of the eleven cases in Class I, Table I, seven died from pulmonary congestion originating at the heart; but in only one of these was there hypertrophy of the right ventricle, and it was joined with great disease of the mitral valve. Of the three who died from sudden stoppage of the heart, there was hypertrophy of both ventricles in two, and of the left only in one. In Case XXXIX, where death arose from disease of the lungs, there was hypertrophy of both ventricles. Hypertrophy of the right ventricle alone occurred in no case in Class II, Table I. In combination with that of the left, it took place in eight cases out of twenty-six, in one of which it was associated with softening. In one there was also mitral disease.

Pulmonary apoplexy existed in eight cases out of the thirty-seven in both classes; of these, hypertrophy of the right ventricle existed in five cases, in one of which extensive mitral disease was also present (XL), and in one aortic disease (L). The other three were LVII, LXI, and LXII. There was excessive pulmonary congestion in many other cases, in only one of which there was hypertrophy of the right ventricle, and in that case there was great softening.

Hypertrophy of the left ventricle occurred in six out of the eleven cases in Class I, Table I, in which there was no dropsy, or other symptoms of general congestion. It was found in eleven cases out of twenty-six in Class II, where signs of general obstruction were manifested; in one connected with softening. In nearly all these cases there was also tricuspid regurgitation.

In Class II, Table I, four persons died from cerebral congestion or apoplexy, viz. Cases XLII, LXV, LXVI, LXVII; there was hypertrophy of the left ventricle in two. In one of the others the ventricles were much softened. In all there was tricuspid regurgitation.

Much the same proportion was observed in the cases which are embodied in Table II. Thus hypertrophy of the right ventricle occurred in thirteen cases out of thirty-nine, in which death resulted from pulmonary congestion, or direct stoppage of the heart, and in thirty-four out of 116, in which it resulted chiefly from anasarca or cerebral congestion; and hypertrophy of the left ventricle occurred in nineteen cases of the former class, and in forty-five of the latter, and, in almost all of these last cases, was associated with tricuspid regurgitation.

So that, on the whole, hypertrophy of the ventricles does not appear to have produced such an amount of arterial congestion in these cases, as to have given rise to any serious consequences.

Attenuation of the walls of the ventricles must of course tend to diminish the force of the circulation, and thus to favour congestion of the venous capillaries. Whether this state, however, is often followed by fatal congestion, or is so frequently associated with such congestions as to induce the belief that it is one of their main causes, is not so easily determined. Of 155 cases in Table II, there was attenuation of the right ventricle in twenty-three, of the left in twelve, and of both in twenty-nine, making in all sixty-four, arising from dilatation without hypertrophy. Of the twenty-three cases of attenuation of the right ventricle, eighteen occurred in Class II, out of 116 cases. Of the twelve cases of attenuation of the left ventricle, three occurred in Class I, out of thirty-nine. All the twenty-nine cases in which both were attenuated, were found in Class II. There was therefore attenuation of the left ventricle in three cases in Class I, and that of the right ventricle in forty-seven in Class II, being respectively in the ratio of 3 : 39, and 47 : 116.

Dilatation, with hypertrophy, was found in the right ventricle in two cases, in both in sixty-two, and in the left alone in ten,—in all seventy-four, twenty of which were in Class I, and forty-four in Class II. So that dilatation and hypertrophy on the right side were as often found coexisting with obstruction of the general circulation as dilatation and attenuation.

Now, as dilatation of the right side was found in almost every case of obstruction of the general circulation, in one half with hypertrophy, and in the other with attenuation, it follows that there is probably some other effect produced on the circulation by dilatation independent of attenuation, which tends to obstruct the return of blood to the heart.

One such effect is an alteration in the shape of the ventricle, whereby the angle of the infundibulum leading to the aortic orifice is enlarged; the blood thus enters this vessel in a more oblique direction, and the streams from opposite points meeting in its mouth are opposed one to the other, and thus the ventricle and afterwards the auricle and veins, become congested. From what we can observe on varying the shape of a vessel from which fluid is flowing, it is probable that no great obstruction is thus offered.

The only other effect on the circulation which can be induced by dilatation must be connected with the valvular apparatus, and to this, therefore, we must look when we examine the alteration which affects each orifice.

The effects of *softening* could not be clearly traced, because it was in some cases joined with and to a certain extent counteracted by hypertrophy.

Valvular derangements—Aortic orifice. There was hardly a single case in which death resulted from obstruction of either circulation where disease of the aortic orifice was the sole lesion. In Cases LIV and LV it had evidently existed a long time without producing any marked inconvenience, until at length the aortic orifice was reduced to a mere slit, the cavities of the heart became mechanically dilated behind it, and with them the mitral and tricuspid orifices were widened to such a degree that their respective valves were unable to close them, and thereby to prevent regurgitation during the systole of the heart. Thus the disease when carried to a great extent, indirectly produced derangement in both circulations.

Dr. Hope has given the particulars of a case in which the aortic orifice was reduced to the size of a pea, without having given rise to any disturbance of the general circulation. Dr. Law,[1] in 1846, presented to the Sheffield Pathological Society the heart of "an out-patient of the infirmary, who died very suddenly whilst waiting to be seen by the surgeon, in consequence of a severe injury to the shoulder-joint some weeks before. He had made no complaint indicating heart disease, excepting having complained of occasional asthmatic attacks of a very slight character. The aortic opening was nearly closed by a complete mass of bone, there being only a small cleft through which the blood could pass. On inquiring afterwards it was found that he had occasionally suffered from fainting fits, but he had never complained of them at the Infirmary." Many other such cases might be adduced. Andral relates a case of extreme narrowing of the mouth of the aorta by calcareous deposits, and states it to be certain that many aged persons are similarly affected without being attacked by dyspnœa or dropsy. He accounts for the absence of these symptoms by supposing the circulation of the aged to be slow. But this state is by no means confined to the aged, as may be seen by reference to Cases LXIX and LXX, nor is the circulation always slow in advanced age.

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[1] Lancet, 1846, p. 566.

I would submit the following explanation which is applicable to all ages. In Cases LIV and LV an unusually protracted rasp-sound was heard commencing with the systole of the heart; which proved that the muscular action of the ventricles was prolonged, by which means a kind of compensation was effected, the blood being thus gradually squeezed through the contracted orifice. This in some degree accounts for the slight effect produced on the circulation by a very considerable amount of aortic obstruction; and as it generally happens that the left ventricle becomes hypertrophied under these circumstances, it is thus enabled to force the blood onward with considerable power.

The same sound here alluded to is not unfrequently heard in aged persons, who yet live on without experiencing any great amount of dyspnœa, and who display no symptoms of dropsy, as in the following case.

Case LXVIII.

Aortic obstruction, without dyspnœa or dropsy.

A lady, æt. 65, was thrown from a carriage, and struck the ground with her left shoulder, in the year 1824. Considerable inflammation ensued. She was attended by Sir P. Crampton, and recovered; but from that time she was subject to violent fits of palpitation, which lasted generally twenty-four hours, in one of which I saw her nine years after the accident. The pulse was intermittent, and there was very lengthened rasp-sound running up the aorta. The diastolic sound was strong, but muffled. On the fit of palpitation subsiding, the intensity of the sound decreased, but its quality remained the same. I saw her again in 1837, when the fits of palpitation were less frequent and less severe, and the pulse less intermittent.

In 1846 I again attended her at Torquay, in consultation with Dr. Battersby and Dr. Toogood. The palpitations had entirely ceased for five years. She was then in her 85th year. The sound was the same as on the two former occasions.

She still lives and is well, having no palpitations, dyspnœa, or œdema of the feet.

Many other instances might be adduced of old persons presenting all the signs of obstruction at the orifice of the aorta, and who are either still living without dyspnœa or dropsy, or else who have died of some other complaint unconnected with the lesion in question; which had hardly been suspected before death, but which was revealed at an inspection of the body.

When the valves are so shortened, or the orifice is so dilated that regurgitation takes place during the systole of the heart, a greater effect is produced on the venous circulation than when obstruction alone is opposed to a current of blood through the aorta. For in this case no increased power in the ventricular walls can prevent the reflux of the blood from the aorta during their diastole, which meeting the current that is flowing from the auricle, offers a great obstacle to it, and thus produces engorgement of the pulmonary veins. But the aperture through which regurgitation takes place from the aorta must be large materially to affect the circulation, because the auriculo-ventricular foramen, through which the blood enters the ventricle during its diastole, is naturally so ample. The following cases illustrate the truth of this remark.

Case LXIX.

Aortic regurgitation—Dilatation of aorta.

A miner, of sallow complexion, æt. 27, had articular rheumatism six years ago, after exposure to wet and cold, but the joints did not swell. He was confined fourteen days by this illness, and has ever since felt a dull, aching pain at the left of the epigastrium, from which he has sometimes been relieved by leeches. He had palpitations for twelve months. His breath was good, and he had walked up to within a day or two of his admission into the Birmingham General Hospital; but for the last fourteen days his epigastric pain and palpitation had increased. There was not the slightest dyspnœa or cough. The impulse of the heart was strong and heaving from a point two inches below the left nipple to the xiphoid cartilage, and even below it and around it. Strong pulsation and purring thrill were felt up the carotid arteries, over each clavicle, and more especially over the top of the sternum, where the aorta seemed to rise up within reach of the finger. Pulsation was also felt under the humeral end of the clavicles, but not under the sternal ends. The pulse was 76, sharp, quick, and strong, generally resilient and double, the second stroke being very feeble. It was very visible at the wrists.

There was no dullness over the usual precordial space; but it commenced two inches below the left nipple, and, like the impulse, extended obliquely inwards and downwards towards the xiphoid cartilage. The respiratory sound was rather feeble, particularly behind the left side. A loud systolic rasp-sound was heard in the course of the carotid arteries, and on the sternum. To the right of the top of the sternum a double rasp-sound was heard, the systolic being the loudest; at the bottom of the right of the sternum it was still heard double, but the diastolic sound was the loudest and the longest. The double sound was also heard at and under the left nipple, but very soft and faint. After a few weeks he left the hospital much relieved. He ceased working in the pits, and set up a horse and cart. He has been seen occasionally during the last three years; the palpitations are less severe and less frequent, but the physical signs remain unaltered.

Case LXX.

Aortic regurgitation.

An iron-roller, æt. 25, at no time very strong, but accustomed to hard work, had enjoyed good health until five years ago, when he began to be troubled with occasional palpitations without cough or dyspnœa. Nine months ago he caught a cold and has had a bad cough ever since, accompanied by expectoration, in general thin, clear, and frothy, at times thicker and more viscous, but still light-coloured. At first he spat a little clear red blood, not amounting altogether to a dessert-spoonful. Two months since a little œdema of the ankles appeared, and the palpitations increased in frequency and severity, and dyspnœa set in, which has been increasing ever since. He has had no pain all along. Five weeks ago he was obliged to desist from work, and although he tried to work again after a little rest, the severity of the palpitations and dyspnœa compelled him to desist. He has lost flesh greatly during the last two months.

On his admission into the Birmingham General Hospital his complexion was yellowish, transparent, and waxy. He had no pain, but felt weak across the loins, and had a dry cough which troubled him much at night. The palpitations were less severe than they had been. There was a trace of œdema about the ankles. His appetite was good, his bowels were open, and his urine was healthy and plentiful. On placing the finger on any of the arteries a sharp, quick blow was given to it, like a drop of water falling in vacuo. They were seen to beat wherever they were superficial. Respiration 30. The pulsations of the heart were visible over the whole of the left side of the chest. A heaving pulsation and a little thrill were felt by the finger pressed down behind the sternum. The sound over the chest was nowhere very clear, but was very dull all around, below, and outside of the precordial region. A fine crepitation was heard behind the lower part of each side. The head was raised by the stroke of the heart against the stethoscope. Two inches below the left nipple, in an oblique outward direction, a systolic, shrill rasp-sound was heard, with a slight trace of diastolic sound. This sound decreased above the nipple, but was heard all up the sternum and to the right of it without any diastolic sound. Under the lower part of the sternum it was double. A loud single rasp-sound was heard behind both sides, particularly near the lower angle of the left scapula.

In a fortnight's time some œdema of the feet came on, but no pulsation of the veins of the neck could be discovered.

He left the hospital and was not seen until very lately, when he was considerably better than at the time he was in the hospital, but the physical signs remained much as they were.

The signs of aortic regurgitation were present in both these cases, and it is further probable that the dyspnœa in Case LXX was caused by mitral regurgitation in addition, and yet both these persons live on in a tolerable state of health.

It therefore appears that a considerable amount of disease of the aortic orifice may exist for a number of years without seriously affecting the health, more particularly if hypertrophy of the left ventricle is joined with it, and that, consequently, this affection has little or no direct influence in retarding the venous circulation and producing engorgement of the vessels of the pulmonary or general circulation.

Orifice of pulmonary artery. The pulmonary artery was considerably dilated in two cases in Class I, Table II, and in two also in Class II, Table II, but always conjoined with other affections.

The valves were much shortened and thickened by the deposition of atheroma and calcareous deposits in three cases in Class I, Table II, in all of which the foramen ovale was open.

There were about six other cases in which these valves were slightly thickened, but not in such a manner as to impair their working.

Disease of this orifice being so rare, and never having occurred singly, it is impossible to estimate its effects on life. The remarks made on the diseases of the aortic orifice equally apply to those of the pulmonary orifice.

Mitral orifice. Out of the thirty-nine cases in Class I, Table II, twelve died suddenly; and of the remaining twenty-seven, who died from pulmonary congestion, there was incompleteness of the mitral valves in twenty-four. It existed in a very marked degree in four cases, and slightly in three others out of eight cases of pulmonary apoplexy, which occurred in Classes I and II, Table I. Of all the forms of valvular disease, therefore, incompleteness of the mitral valves was found to be the most constant and direct cause of pulmonary congestion.

Simple mitral obstruction, unless it be considerable, and unless the left ventricle be also attenuated or softened, is overcome by the force of the ventricular diastole, in the manner indicated in discussing the effects of hypertrophy. Accordingly such a state may exist for a considerable time without giving rise to a fatal termination, as may be seen on reference to Cases XXXIV, XXXV, XXXVI, LIII, and others. Mr. Prescott Hewitt[1] described a case to the members of the Medico-Chirurgical Society of London in 1846, in which, during lifetime, no symptom of diseased heart was observed, and after death the mitral orifice was found nearly closed; there being merely an aperture the size of a quill for the passage of the blood. This state of the mitral valve is often overlooked; because, as was observed by Dr. Hope, if the contraction is extreme, the current is too small to engender sound; and if it is but slight, sufficient space is left for the blood to pass from the auricle into the ventricle without such an amount of friction as to occasion murmur.

Mitral regurgitation, however, is a much more formidable affection, because the power of the ventricle during its systole increases the force with which the blood regurgitates into the auricle. It existed in almost every one of the cases where pulmonary congestion formed the leading symptom, both in Class I and II. It may arise from disease of the valves, dilatation of the orifice, or from both causes. In Table I it arose from disease of the valves alone in six cases, and combined with dilatation in four; from dilatation alone in none: there were two such cases, however, in Table II. Since, therefore, four cases only of mitral dilatation have been recorded as occurring out of twenty-eight cases of dilatation of the left ventricle, it would appear that dilatation of the left ventricle does not necessarily or even frequently cause dilatation of the mitral orifice. It should be mentioned, however, that in five other cases the orifice was dilated, but that regurgitation did not take place to any great extent, in consequence of the large size of the valves (Cases LIV, LV, LVII, LXI, and LXII), and this occurred in Table II fourteen times. In the great majority of cases, the mitral orifice was contracted by disease, whilst the ventricle and auricle were dilated. And even in certain cases where there was no positive disease, the mitral zone was found very strong, thick, and unyielding. Simple dilatation, therefore, of the mitral orifice, to such an extent as to admit of regurgitation, was very rarely met with in these cases.

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[1] Lancet, 1846, p. 5.

The mean circumference of this orifice is estimated by Bizot at four inches, being half an inch less than the tricuspid orifice. In general the two valves can more than close this space, so that a certain amount of dilatation may exist without regurgitation taking place. The relative sizes of the valves and orifice were not so accurately measured as that of the tricuspid valves and aperture; consequently, some slight incompleteness may have been overlooked.

As the mitral valves were inefficient in about one third of the cases in Class II, this affection might be supposed to be productive of obstruction of the general circulation, and so it doubtless is indirectly. It will presently, however, be seen, that in nearly every one of these cases there existed a direct obstruction to the blood entering the right ventricle, from the auricle and veins, which must of course have superseded any obstruction on the left side of the heart.

Tricuspid orifice. Atheromatous and calcareous deposits were scarcely ever met with in this situation, and consequently obstruction was seldom found.

Regurgitation, as in the case of the mitral aperture, was induced either by disease or by simple dilatation.

In every case except those wherein the foramen ovale was open, the changes which impaired the efficiency of the valvular apparatus were such as prevented the valves from closing the orifice, and thus permitted regurgitation.

The valves were either corrugated, adherent to the walls of the right ventricle, or their cords were shortened. Very rarely the valves were both attenuated and shortened; generally, when they were shortened, they were also thickened and shrivelled. Sometimes one valve adhered closely to the side of the ventricle, and no trace of any lymph, old or recent, could be found; but in others, as Case LIX, milky patches were seen on the lining membrane of the right ventricle, close to the adhesion, which were evidently of inflammatory origin. The tricuspid valves were diseased in five cases, in Class I, Table II, and in forty-six cases, in Class II. In three of the five cases in Class I, the foramen ovale was open, and in the other two the valves were only fringed with vegetations, and were slightly shrivelled.

The forty-six cases in Class II were thus made up. The principal disease was adhesion of one valve in ten cases; shortened chordæ tendineæ in twenty cases, and thickened and shrivelled valves in sixteen cases. Besides these changes, on some of the valves thus affected vegetations were found, and in other cases the valves were attenuated and perforated; in some, again, one of the cords had apparently been broken, and was curled up into a small nodule like a pin's head; but this last affection would probably not much affect the working of the valve, for I have found it in the bodies of persons who had never presented any traces of heart disease, or obstruction of the circulation.

Since the tricuspid valves were diseased in fifty-one cases, and the mitral valves in fifty-three, it follows that, contrary to the generally received opinion, the one orifice is as liable to disease as the other. But the disease of the mitral orifice is generally palpable, while that of the tricuspid valves may very often escape the eye of a superficial observer. And yet the shortening of the cords of the tricuspid valves, so easily overlooked, would probably, for reasons before stated, have a much more serious effect on the circulation than an obvious thickening and narrowing of the mitral orifice, or a deposition of vegetations on the fringes of its valves.

It must be remembered, however, that this amount of tricuspid disease was found in persons all of whom during lifetime had exhibited unequivocal signs of cardiac derangements, and in the greater number of whom death took place from obstruction of the pulmonary or general circulation. But were I to add to these cases all those in which death resulted from other causes, yet in which there were also diseased cardiac valves, then the proportion would be greatly altered, and the number of cases, in which the valves on the left side of the heart were diseased, would more than treble the number of those in which the valves on the right side were affected.

When the tricuspid orifice was dilated beyond its normal size, it was in nine tenths of the cases connected with dilatation of the right ventricle and auricle. Thus, contrary to what took place on the left side of the heart, the tricuspid orifice was almost always dilated simultaneously with these two cavities.

This may readily be accounted for. The dilatation of the right cavities was generally carried to a much greater extent than that of the left cavities, although I am not prepared to give the exact relative admeasurements, from the difficulty experienced in taking them accurately. The fibrous zone of the tricuspid valve was less frequently affected with disease tending to thicken and harden it than was the mitral zone; the inflammatory action which attacked the former generally tending to diminish the cohesion of its parts, and to dispose it to dilate.

Whatever, however, were the reasons, dilatation of the tricuspid orifice was found in 91 out of 105 cases of dilatation of the right ventricle, and in 10 cases without that of the latter. In 46 cases it was also joined with a diseased state of the valves.

It was not noted unless the circumference of the orifice exceeded five inches in the male, and four inches and a half in the female. Bouillaud makes its average circumference in the male four inches; Bizot four inches and a half. I place the greatest possible reliance on the accuracy of Bizot's investigations; but my own measurements would have led me to estimate the circumference at about four inches and a quarter. The measurements, however, on which this opinion is based were all made in the Birmingham General Hospital, on subjects which were, for the most part, furnished from the ranks of the manufacturing classes, who are not generally endowed with vigorous constitutions. This may possibly account for the difference in the results of my measurements from those of Bizot. To cover the area of a circle, whose circumference varies from four inches to four inches and a half, each segment should have a height of eight or nine lines; if they are unequally sized, the joint height of two, or the diameter of the circle should be from sixteen to eighteen lines, or thereabouts. If the tricuspid orifice be dilated, so that its circumference reaches six inches, each valvular segment should be about one inch in height. If they are less than this, a space is left open in the middle of the orifice, through which the blood can regurgitate during the systole of the heart. Some allowance must be made for the diminution of the aperture by contraction of the base of the ventricle, but this probably does not take place to any great extent.

Hunter, and subsequently Dr. Adams and Mr. T. Wilkinson King, have asserted that in health the tricuspid valves seldom completely close the orifice to which they are attached. The latter gentleman found that when fluid was forced into the right ventricle from the pulmonary artery it generally found its way through the tricuspid orifice into the auricle. I have frequently repeated this experiment with the same result, unless I pinched in the base of the right ventricle with the hand, when the regurgitation was frequently prevented.

If, therefore, the valves scarcely close the orifice in health, it is clear that when it is dilated, regurgitation must take place during the systole of the heart, unless the valves are also proportionately increased in size. This took place in fifteen cases out of eighteen in which the right ventricle was dilated, in conjunction with the tricuspid orifice, in Class I, and consequently there was no regurgitation. Thus the valves were increased in size in fifteen cases only out of 121 in which the orifice was dilated; a very different result from that which Dr. Hope supposed took place, as he conjectured that the valves generally increased in size with the dilatation of the orifice, and that consequently such dilatation did not induce obstruction to the general circulation by means of tricuspid regurgitation.

Mr. T. Wilkinson King, arguing from the fact of a certain amount of regurgitation often taking place during health, assumed that the tricuspid orifice in this manner acted as a kind of safety-valve, and thus prevented too much blood being thrown into the pulmonary vessels. This view is founded on the belief that such active congestion is easily induced, and is very serious in its effects. It has been shown, however, to be probable that this action of the ventricles has been overrated; and it will presently be seen that it would not improve the chances of life, did nature thus attempt to relieve arterial congestion by means calculated to induce venous engorgement.

When regurgitation takes place to any extent, it is impossible to conceive a more powerful obstruction than is thus offered to the passage of the blood from the veins into the heart, by a strong counter-current forced backwards by the systole of the right ventricle.

Tricuspid regurgitation took place in four cases out of thirty-nine in Class I. In three of these the foramen ovale was open, so that the full force of regurgitation was not sustained by the general venous current alone; but as the blood also entered the left auricle through the foramen ovale, its effects were felt in part by the pulmonary veins, and thus were divided between the two venous circulations. On this account probably the obstruction to the general circulation was insufficient to give rise to anasarca; and therefore these were exceptional cases in appearance rather than in reality.

There only remains then one other case in which tricuspid regurgitation was not attended by disturbance of the general circulation. It will be remembered that in Class I there were fifteen cases in which the tricuspid orifice was dilated, but the valves had proportionately increased in size. Cases XXXIV, XXXVII, XXXVIII, and XXXIX were of this description.

On the other hand, in Class II there was tricuspid regurgitation in 106 cases, leaving only ten exceptional cases in which obstruction of the general circulation was not preceded by this occurrence.

An examination of thirty-four cases of cardiac dropsy, recorded by Bouillaud, shows that tricuspid regurgitation took place in twenty-two of them, and that in seven others the right ventricle and auricle were both dilated; whence, from what we have seen in the above cases, we may reasonably infer that the intermediate orifice was also dilated, although no express mention is made of it, and that consequently regurgitation took place in them also. In two the right ventricle was stated to have been much contracted, by which means obstruction would be promoted. So that out of thirty-four cases there were only three in which there was not either tricuspid regurgitation or obstruction, a proportion very similar to that observed in my own cases.

It is impossible that these results can be accidental, nor can it be said that the incompleteness of closure has been exaggerated; for had this been the case in one class it would have shown itself in the other, and we should have had some cases of imperfect closure of the tricuspid orifice occurring in Class I.

Here, then, we have the solution of the difficulty with which we started. Dilatation is the main cause of general obstruction, not, as supposed by Dr. Hope, because the walls of the ventricle became attenuated, for in fact they were as often hypertrophied as attenuated; nor yet, as supposed by Andral, because there "is an excess of the capacity of the heart relative to that which has been preserved in the blood-vessels," but because it is accompanied by incompleteness of the tricuspid valve, in consequence of which a powerful back current is forced against the blood returning from the veins of the general circulation.

It is probable, also, that this impediment to the circulation exists in many other cases which do not terminate fatally. It has been customary to attribute the anasarca that appears after great depletion, fever, and other debilitating causes, to weakness of the heart alone. It is possible, however, that, in consequence of this weakness of the muscular walls of the ventricles, they become dilated, and with them the tricuspid orifice. In many of these cases I have seen the anasarca preceded by strong venous pulsations in the neck. On the renewal of health and strength this dilatation may subside, and the causes of obstruction cease.

It is unnecessary here to trace out the effects which may be produced on the different organs by this powerful cause of impediment to the venous circulation. It can easily be imagined that many diseases may thus be induced. Indeed, when it is considered that dilatation of the heart is so often joined with hypertrophy, it is more reasonable, after what has been seen, to attribute the greater number of apoplectic attacks which occur in connexion with diseased heart, as much or more to tricuspid regurgitation than to hypertrophy of the left ventricle.

The termination of such cases must greatly depend upon the presence or absence of structural changes of the valves. In some cases all the symptoms of engorgement will disappear under suitable treatment.

CASE LXXI.

Feeble heart—Venous pulsations in the neck—Dropsy—Recovery.

A miner, æt. 66, had been troubled with a cough and shortness of breathing for a year or two, with occasional palpitations. Three weeks since, his legs, and subsequently his thighs and scrotum, swelled. On his admission into the Birmingham General Hospital, his urine was scanty, 1024 sp. gr., and contained no albumen. His tongue was clean, and his appetite was tolerably good. The pulse was under 100, soft and feeble, but regular. The veins of the right side of the neck were large, tortuous, and undulated. The respiratory sound was rather feeble. In different parts of the chest, particularly at the posterior parts, bass-viol and cooing sounds were heard, with occasional large, dry crackling.

The heart was seen to beat two inches below the left nipple towards the outer side of the chest, and also at the xiphoid cartilage. At the former spot a soft and rather prolonged bellows-sound accompanied the systole of the heart, diminishing in intensity in every direction. Under the use of tonic and diuretic medicines, and a generous, full diet, the anasarca gradually disappeared, as did the engorgement and undulations of the veins in the neck, and in less than eight weeks he left the hospital, feeling better than he had done for years.

Case LXXII.

Venous pulsations—Dropsy—Recovery.

A gentleman, æt. 68, of nervous temperament, had enjoyed good health until within two or three years, and during that time he had applied for advice whilst suffering from influenza. An examination of the chest after his recovery from this attack showed that he laboured under chronic bronchitis with some emphysema. On this occasion dyspnœa was most urgent. He had œdema of the legs and feet. There were strong pulsations in the external jugular veins. His pulse was full and hard. There was slight fluctuation in the abdomen. The respiratory sound was feeble, and mixed with cooing and bass-viol sounds, with here and there some small mucous rattle. At the bottom of each side it was absent, and there was dullness on percussion. The action of the heart was heaving. An obscure systolic bellows-sound was heard a little to the left of the lower third of the sternum, but not extending in any direction. Simple diuretic medicines produced no effect, but under the use of infusion of digitalis with ammonia he rapidly recovered; the anasarca, dyspnœa, and venous pulsations all disappearing.

Eighteen months after this he had a similar attack, but of a more obstinate nature, his legs being much swelled and very red; and he complained greatly of their itching. The bellows-sound again appeared. After the lapse of some time, and the employment of elaterium, digitalis, &c., the anasarca left him. He has continued in his usual state of health for the last two years.

CASE LXXIII.

Venous pulsations—Dropsy—Recovery.

A lady, æt. 43, was married at the age of thirty, but has had no family. Previous to her marriage she was very pale, and was subject to palpitations, dyspnœa, and swelling of the ankles. Menstruation was, at that period, very scanty and irregular. Since her marriage a slight improvement has taken place in this respect, but at times she suffers in an exactly similar manner. For the last four months she has menstruated very slightly, and has suffered much from flatulence and other dyspeptic symptoms. In this state she applied for advice. The sounds of the heart were feeble, but clear, and were heard over every part of the chest. The veins of the neck were slightly swollen. She derived much benefit from tonic and alterative medicines, which in a great measure removed the dyspepsia. In a few months' time she returned, with œdema of the legs and thighs, and a trace of ascites. When she was seen in consultation with Mr. Baynham, the dyspnœa and palpitations were oppressive. The most active diuretic medicines had been employed without the least benefit. The veins of the neck were now seen to be very much more swollen, and a pulsation in them was manifest. The urine was not coagulable by heat.

A small quantity of blood was taken from the arm, and tonics were combined with diuretics. In less than three weeks the swelling and pulsation of the veins subsided, and every trace of dropsy disappeared.

The cause of dilatation in all these cases was most probably subacute bronchitis supervening on chronic bronchitis. Indeed I had on several occasions attended the subject of Case

LXXII for such attacks. It is probable that many cases of dropsy from bronchitis, as they are commonly called, are thus produced.

Case LXXIV.

Mitral disease—Venous pulsations—Dropsy—Relief.

A tradesman's daughter, æt. 15, had a severe attack of rheumatic fever, which left her troubled with dyspnœa and palpitations. Dropsy having come on, and not being speedily relieved, her parents brought her to me for advice.

The action of the heart was feeble, and the pulse very small. The veins of the neck were swollen, and pulsated slightly. Undulations were seen between the second and third left ribs, and below the nipple. The precordial dullness extended upwards. A strong and rather coarse bellows-sound was heard below the left nipple, and also near the lower angle of the left scapula, seeming in the latter situation to be close to the ear. Cooing sounds were heard in different parts of the chest. She was relieved, and the anasarca disappeared, the veins of the neck remaining rather full. Some months after this the anasarca returned. Seen in consultation with Mr. Pye Chavasse, the dyspnœa was so great as to threaten suffocation, and the legs and abdomen were amazingly distended. The strongest diuretic medicines failed to act on the kidneys, nor was any relief obtained from various remedies. It was proposed to employ acupuncturation on the thighs, but objected to by the parents. In a few days' time large bullæ rose on the legs and burst, the sores putting on a black gangrenous aspect. Much serum ran from them; and in a few day's more the anasarca had greatly diminished, and the sores on the legs assumed a healthy appearance. The dyspnœa was much relieved, and the kidneys resumed their functions. In a word, the patient gradually recovered her ordinary health, and is now alive; but the physical signs of valvular disease remain the same, and she cannot do any work.

This is indeed a remarkable case. After witnessing it, who would ever despair of a favorable termination in cardiac dropsy? The physical signs of dilatation of the left auricle and of mitral regurgitation could not be mistaken. Here, therefore, was a temporary recovery from dropsy, although mitral regurgitation existed and still remains. The immediate cause being recognised, it is therefore possible that this formidable sequela of heart disease may not unfrequently be temporarily arrested, although the indirect causes may be altogether beyond reach.

From the analysis of Bouillaud's cases, it might be supposed that he was fully aware of the connexion between tricuspid regurgitation and obstruction of the general circulation. He remarks, that "the dilatation of the orifices of the heart is not less common than that of the cavities themselves," and recommends diligent observers not "to neglect examining the orifices of the heart in all its organic diseases; for if the dimensions are such that the valves cannot close the orifices, the circulation of blood through the heart must be more or less seriously disturbed." Throughout the whole of his work, however, he attributes the disturbances of the general circulation to disease of the valves on the left side of the heart, and seems to be totally unacquainted with the fact here elicited, that tricuspid regurgitation almost invariably precedes the appearance of dropsy, whether the valves on the left side of the heart be diseased or not. Dr. Hope goes further than this, by attributing dropsy to dilatation, by which the walls of the ventricles are attenuated and weakened, so that the circulation becomes languid, and the blood stagnates more or less in the veins. But he expressly states that tricuspid regurgitation is very rare.

Authors in general, allowing dilatation of the tricuspid orifice to be a cause of regurgitation, and consequently of obstruction to the general circulation, seem to think that it seldom takes place, and have very often overlooked both the extent of the dilatation and the diseases of the valves. These latter, indeed, although most formidable in their effects, are by no means of such a nature as prominently to meet the eye. Whatever, indeed, may be the views of any particular writer, the existence of tricuspid regurgitation in almost every case of obstruction of the vessels of the general circulation originating at the heart, does not appear to be known to or received by the profession at large.

The following conclusions seem to result from the facts detailed in this chapter.

1. A considerable amount of obstruction may exist at the aortic orifice of the heart, without seriously affecting the general health.

2. Mitral regurgitation is one of the most direct and frequent causes of pulmonary venous congestion.

3. Tricuspid regurgitation is the most direct and almost *constant* cause of that engorgement of the vessels of the general circulation, and its consequences, which originate with the heart.

4. Except in conjunction with regurgitation through the auriculo-ventricular orifices, hypertrophy of the ventricles in many cases rather assists the circulation than promotes congestion.

CHAPTER XII.

DIAGNOSIS OF CHRONIC HEART DISEASES.

When we meet with persons labouring under palpitation, dyspnœa, irregular pulse, fluttering or constriction at the precordial region, and other symptoms indicative of derangement of the action of the heart, the first point to be determined is whether this derangement depends on *organic* or *inorganic* causes.

Inorganic causes. The principal derangements of the action of the heart which arise from these causes are—

1. Dyspepsia. 2. Hysteria and nervous irritability, from whatever source derived. 3. Hyperemia. 4. Anemia.

When evidences of one or more of these states exist, we cannot from this conclude that inorganic causes alone are present, because organic changes of the heart may also exist at the same time. Before we can arrive at such a conclusion, we must ascertain that no signs of these latter affections exist. This is by no means difficult in respect to dyspepsia, nervous irritability, and hyperemia. In anemia, however, a murmur is sometimes heard of the same character as that which occurs in aortic obstruction. It is seldom, however, persistent during a state of perfect repose; for instance, during sleep. In these cases a somewhat similar sound can generally be made to appear by gentle pressure on the course of the arteries of the neck, and a musical hum may by the same means be produced in the large veins which approach the surface of the body. So that when these latter sounds are heard, and the aortic murmur does not exceed that of a soft bellows-sound, we may safely conclude that the main causes of derangement are inorganic.

Occasionally, however, when the debility and irritability are extreme, the heart becomes dilated to such an extent as to lead to tricuspid and mitral regurgitation. In that case, signs presently to be detailed will reveal this state with tolerable accuracy.

Organic causes. If, however, it be decided that some organic changes have taken place in the heart itself, the next point to be determined relates to the nature of such changes; whether the contractile power of the heart, or its valvular apparatus, or both are affected; and, if the contractile power be affected, whether it is increased or diminished.

Increase of contractile power. If this be constant, it must arise from hypertrophy of the ventricle, and if it be very great, the impulse communicated by the heart to the hand or head laid on the chest will be considerable, and will have more or less of a heaving character; the extent of the precordial dullness also will be increased, more particularly if, at the same time, the ventricles are dilated. The systolic sound, too, will be more muffled, as has been explained in the remarks on the formation of the sounds of the heart. In hypertrophy by extent, however, the quality of the systolic sound may be unaffected, whilst its intensity is increased, and it is heard at a greater distance than usual from the precordial region.

Decrease of contractile power. This must arise from attenuation and softening, and possibly from adhesion of the pericardium to the heart. Whether the heart be dilated or softened, the impulse is diminished.

When simple attenuation from dilatation exists, the extent of dullness over the precordial region is increased, and the sounds of the heart, although feeble, are sharp and clear, and are heard to a much greater distance than usual from the spot where they are engendered. When the ventricles are softened, especially if they are at the same time hypertrophied, both sounds are extremely feeble, and very muffled and confused. A softened state of the heart may often be thus recognised.

Adhesion of the pericardium. Dr. Saunders thought that this state might be discovered by a pulsation, or rather a retraction in the epigastric region, synchronous with the systole of the ventricles. The pericardium adhered to the whole circumference of the heart in six of the cases here recorded, and in another it was partially adherent, but in the last alone was this sign observed.

Pulsations were occasionally observed at the outer part of the left side, generally between the seventh and eighth ribs, which depended on adhesion between the pericardium and side; but in one case the pericardium did not adhere to the heart, and in Case XXXV it was connected with the side by long bands. I have seen this sign make its appearance in several cases after rheumatic fever, with pericarditis, two of which occurred in the wards of St. Thomas's Hospital in 1833, and on one or two occasions after pleuritis, but I have not had an opportunity of examining the bodies of these persons.

Dr. Hope has described a kind of jogging action of the heart as characteristic of this affection. I have long sought for this sign, but never could find it, nor have some other observers been more successful. The action of the heart was quite different in some from that which was observed in others of the cases above alluded to. In one, Case XXXIII, it was strong and quick as the stroke of a hammer; in another, Case XLIV, very feeble and irregular; in others very moderate.

I am, consequently, unable to mention any sign by which the adhesion of the pericardium to the heart can be recognised.

If *derangement of the valvular apparatus* is suspected, we have to determine its *seat* and *nature*. Here our attention is at once directed to certain orifices of the heart by the general signs present. From what we have seen of the progress and termination of these diseases, if there be neither urgent dyspnœa, nor any signs of obstructed general circulation, we shall suspect the aortic orifice; if very urgent dyspnœa be present alone, then we shall look to the mitral orifice, in expectation of finding regurgitation taking place through it. If there be signs of obstruction to the general circulation, we shall expect to find tricuspid regurgitation, with or without disease on the left side of the heart, according as the signs of pulmonary obstruction are present or not.

We will now then proceed to examine in succession the signs of derangement of the valvular apparatus at each of these orifices, omitting all consideration of those which affect the pulmonary artery, as being of little practical value, owing to their extreme rarity.

Aortic orifice. Some writers have asserted that the seat of valvular murmurs is clearly indicated by their being heard with the greatest intensity at the exact spot on the surface of the chest which immediately lies over the orifices in which they are respectively engendered. Thus disease of the aortic orifice would give rise to a murmur, the greatest intensity of which would be under the left edge of the sternum below the third rib. Very frequently, however, the maximum of such sound is heard considerably to the right of this point, as in Case LIV and others. This I have generally found to occur in cases of hypertrophy. I have frequently found it impossible to ascertain the seat of murmur from the spot in which it was most distinctly heard in lifetime.

Dr. Williams has drawn attention to the direction in which murmurs are propagated, and this has seemed to me to furnish much more valuable information. Thus when the murmur is aortic, it can generally be clearly traced for a considerable distance up the course of the aorta, and in certain cases it is heard louder towards the arch of the vessel than over the seat of the aortic valves, as has been shown in the remarks on aneurism. When the disease is obstructive only, the murmur is systolic, and its strength and quality of sound depend in a great measure on the contractile power of the left ventricle, and on the nature of the material with which the orifice of the vessel is obstructed.

It has been seen that, in certain cases of aortic obstruction, the murmur was very prolonged, as in Cases LIV and LV. It was never found to possess this character unless aortic obstruction was present, and unless also the orifice was very much contracted.

When aortic regurgitation takes place, the sound is either diastolic or double. The diastolic sound in such cases is often heard further to the right side than is the systolic sound.

In all cases of aortic regurgitation here recorded, there were visible pulsations of the arteries, to which attention has been directed by Dr. Corrigan. They were also witnessed in certain cases of hypertrophy, such as Case XXXVII, and others, without any regurgitation whatever; so that we cannot infer the existence of aortic regurgitation from this sign alone, unless we have clearly ascertained that the heart is not hypertrophied.

Mitral orifice. The physical signs of mitral obstruction are not in general so well marked as those of aortic obstruction; in a great measure, because the size of the auriculo-ventricular orifice is so large, that a considerable amount of disease may exist without sufficiently narrowing it to give rise to sound. When a murmur is thus produced, it is of course diastolic; and, as a general rule, it may be heard louder towards the apex of the heart than elsewhere, and also with great distinctness near the lower angle of the left scapula. It is often, however, extremely difficult by its seat alone to distinguish this murmur from that of aortic regurgitation; but, as in the latter case, there would be visible arterial pulsations, and none in the former, the differential diagnosis is rendered tolerably sure. Some also would insist on the difference in the pulse in these two cases; it being small, irregular, and tremulous in mitral disease, and jerking and sharp in aortic regurgitation. The value of such a sign may, however, be fairly questioned, for the reasons which have been previously stated in the chapter on the Diagnosis of Aneurism. It is certain that a very great amount of obstruction may exist at the mitral valve in some cases where, in consequence of the aperture being contracted to a very narrow slit, the stream passes noiselessly through it; such cases, however, are rare.

Dr. Hope seems to take it for granted, that mitral regurgitation is always accompanied by a systolic murmur. In Cases XXXII, XLIX, and others, in which regurgitation must have taken place through the mitral orifice, no trace of murmur was detected, and the cause of its absence has been explained when the formation of murmurs was discussed in Chapter III.

In two or three cases, a systolic murmur was heard chiefly towards the apex of the heart; and, on inspection, the mitral valve was found complete. In two cases of this kind, of which Case LIII was one, the ventricular sides of the valves were covered with rough vegetations, so that probably the murmur was caused by the blood passing over this roughened surface. It is difficult to see how such a sound as this could be distinguished from that produced by mitral regurgitation.

It would appear, therefore, that it is not always possible to detect mitral regurgitation by a murmur when it exists, and that not unfrequently no murmur at all is engendered.

There was a sign of mitral obstruction or regurgitation when joined with dilatation of the left auricle, which was observed in some of these cases, and which I am not aware has been noticed elsewhere. A faint pulsation or undulation was seen between the second and third left ribs, and it was often difficult to determine whether or not it was synchronous with the systole of the ventricles. It was present in Case LI and others. On an inspection of the bodies, the left auricle was found greatly dilated, its appendix was uncovered by lung, and was in direct contact with the walls of the chest.

Tricuspid orifice. Obstruction so rarely occurs at the tricuspid orifice that it is unnecessary to inquire into the means of ascertaining its presence.

It has been generally assumed that when regurgitation takes place in this situation a systolic murmur is produced. So far, however, was this from being the fact in the cases here recorded, that a murmur was very seldom heard which could be traced to this source. Thus in Cases XLIX, LIX, LX, and others, where considerable regurgitation took place, no murmur whatever was heard.

There is, however, a very valuable sign of tricuspid regurgitation which is frequently found, and which is furnished by venous pulsations or undulations in the neck. These are generally much more marked on the right than on the left side. Dr. Hope, however, thinks they very often arise from hypertrophy alone; but they were only seen in three cases unaccompanied by regurgitation. If they often arise from hypertrophy alone, there would surely have been some traces of them in Case XXXVII, and others of a similar kind.

On the other hand, however, the absence of venous pulsations by no means proves the absence of regurgitation, for in several cases of decided regurgitation, in which the heart was either attenuated or softened, there were no venous pulsations. In fact, venous pulsations, although they raise a strong presumption in favour of the existence of regurgitation when they are present, are by no means a measure of the amount of the obstruction which is offered to the circulation by such regurgitation. Their strength is rather a measure of the contractile force of the heart. Thus if both ventricles are hypertrophied a strong venous current will be met by an equally strong regurgitating current, the shock will be great, and the pulsations up the veins of the neck will be strongly marked. If both ventricles are softened or attenuated, a feeble venous current will be met by an equally feeble regurgitating current, and the thrill and pulsation will be slight, whilst the flow of venous blood will be as much impeded in the one case as in the other.

From what we have seen of the progress and termination of these cases, the existence of general dropsy, not accounted for on any other supposition than obstruction at the heart, would render the existence of tricuspid regurgitation extremely probable.

We have, then, the following signs of valvular derangements:

AORTIC ORIFICE—*Obstruction.* Systolic murmur traced up the course of the aorta, sometimes very prolonged.

Regurgitation. Diastolic murmur running up the aorta—visible arterial pulsations.

MITRAL ORIFICE—*Obstruction.* Sometimes diastolic murmur at the apex of the heart and at the lower angle of the left scapula, not up the aorta—without visible arterial pulsations. Pulmonary obstruction.

Regurgitation. Sometimes, but not often, systolic murmur, heard at the apex of the heart, and at the lower angle of the left scapula. Occasionally undulations between the second and third left ribs. Pulmonary obstruction.

TRICUSPID ORIFICE. *Regurgitation.* Seldom any murmur. Venous pulsations of the neck. Obstruction of the general circulation.

If these views are correct, it follows that cases must not unfrequently occur in which it is difficult, if not impossible, to determine the exact seat or amount of valvular derangement, and that, consequently, the refinements in diagnosis of some writers cannot be altogether carried out at the bedside. Happily this is of less practical importance than would be imagined at first sight. Correctness of diagnosis is valuable only as it lays a foundation for treatment. With this object in view, the chief endeavour of practical men will be to determine if organic disease of the heart is present, whether its contractile power is increased or decreased, and whether either or both of the circulations which result from it are obstructed. This will be accomplished in most cases with ease, although it may not be always possible to predict the exact state of each of the different valves.

CHAPTER XIII.

TREATMENT OF CHRONIC HEART DISEASES.

Systematic writers, following the classification they are compelled to adopt, have laid down the treatment which they have deemed respectively suitable for valvular disease, hypertrophy, dilatation, and other chronic affections of the heart. But, as these different states have been shown to be often complicated the one with the other, it may be practically useful to consider their treatment in reference to the different stages which have been seen to occur in the progress of these diseases in general, and through which they reach a fatal termination.

These stages have been shown to be characterized by the absence or presence of symptoms of obstruction of the pulmonary or the general circulation.

Before, however, entering on a consideration of the treatment of these affections, after they have appeared, it may be advisable to offer a few remarks on that which is calculated to prevent their formation.

A certain number of cases of valvular disease, hypertrophy, and dilatation with attenuation, result from inflammatory attacks. Some of these, as acute pericarditis, are well marked, and therefore are generally recognised. If such cases were treated, on the plan detailed in the chapter on Pleuritis, with mercurial frictions and leeching, rather than by copious venesection, I cannot but think that the consequences would be in general less serious than they usually are, that the heart would less frequently adhere to the pericardium, and that hypertrophy, dilatation, and valvular disease would be less common.

There are other forms of inflammation, of a low subacute character, which are most insidious in their approaches, and therefore very difficult of discovery. The manner in which pericarditis and endocarditis thus occur in rheumatism is so well known, that a careful practitioner is always on the look-out for them. As a precautionary measure, it is useful to rub in some mercurial ointment over the precordial region in all cases of acute articular rheumatism, whether there are physical signs of pericarditis and endocarditis or not.

There are two other forms, however, in which this dangerous disease may be very frequently overlooked. The one is when it occurs after accidents or operations, and is illustrated by the following cases.

Case LXXV.

Endocarditis and pericarditis after amputation of the leg.

A boy, æt. 5, was received into the Birmingham General Hospital with extensive laceration of the right leg, which had been caused by a waggon-wheel passing over it. The tibia was denuded, and the superficial and deep-seated muscles of the calf were torn from each other. He was restless in the night, and early the next morning he was seized with convulsions, alternating with rigid extension of the trunk and limbs. The pupils were dilated and insensible to the action of light. He was unconscious, but occasionally cried out. His pulse was very feeble and so frequent, that it was difficult to count it. At 10 a. m. the limb was removed by Mr. Hodgson, apparently without the boy being sensible of the operation. The convulsions and spasms ceased in about four hours, and never returned.

He improved rapidly for three weeks, when he was found one day feverish with flushed face, hot skin, and white tongue. The pulse was quick in its stroke, from 90 to 100. He was fretful and had a slight cough, and the respiration was accelerated. On examining the chest, a little cooing sound was heard here and there, and the action of the heart was quick and jerking. The next day he was still flushed and feverish, and had an anxious expression of countenance. The respiration was more embarrassed. His cough was frequent and dry. The pulse was 100, full, hard, and jerking. The precordial region was dull on percussion up to the third rib. A coarse bellows-sound was heard below the left nipple and up the course of the aorta, but was not heard at the back. Four leeches were immediately applied over the region of the heart; some calomel and Dover's powder were administered, and strong mercurial ointment was rubbed into the precordial region. The next day all his symptoms improved. There was still much cooing sound. He gradually but completely recovered.

It would be out of place to comment on the extremely interesting points of this case in reference to its surgical treatment, any more than to remark that the state of the nervous system seemed to demand rather than to counter-indicate the operation; for by this means the main source of irritation would be removed, and the reflex action controlled. The discovery of pericarditis was unexpected, and shows the necessity for carefully examining the chest for a long time after accidents and operations, since pericarditis is not the only affection which thus insidiously creeps on and destroys the patient.

Case LXXVI.

Pericarditis after amputation of the leg.

A fine healthy lad, æt. 15, whilst employed on the Birmingham and Derby railway, was thrown down, and a heavily loaded waggon passed over his right leg and thigh. On being brought to the Birmingham General Hospital it was found that he had received a severe compound fracture of the leg, with great laceration of the soft parts, and injury to the knee-joint. In about half an hour the limb was removed by Mr. Hodgson above the knee.

On the third day after the operation he felt a pain at his heart, with dyspnœa and "catching" of his breath. The pulse was 130, hard and small, the skin was dry and hot, the urine high coloured and scanty. The whole of the abdomen was rather tender, and the precordial region very painful on pressure. The respiration was hurried. The action of the heart was quick, but not strong. There was no precordial dullness; but a creaking sound was distinctly heard two inches below the left nipple towards the sternum, but not above it, which persisted during the suspension of respiration. A soft, systolic bellows-sound was heard chiefly to the left of the sternum, between the second and third ribs. Eighteen leeches were applied, with strong mercurial frictions over the precordial region, and calomel and opium were given. The next day the creaking sound was heard higher up the chest, but still near the sternum. The bellows-sound was also heard all up the course of the aorta; but the precordial pain had left him. In three days more the abnormal sounds at the heart disappeared, with the exception of a slight prolongation of the systolic sound. He recovered completely.

The general signs of pericarditis were less obscure in this case, but still were by no means strongly marked.

These two cases were examined at the request of my friend and colleague Mr. Hodgson, and the notes are published with his approbation.

The other form in which obscurely marked inflammation of the membranes of the heart occurs, is one which often puzzles the most experienced practitioners. The patient complains of some slight ailment, it may be in the head, chest, or abdomen, and is treated accordingly; but, on the relief of such symptoms, the pulse remains very frequent, and has a little sharpness about it. The sounds of the heart are seldom affected, except that sometimes the diastolic sound is unusually sharp. Phthisis is often, and with just cause, suspected; but no physical signs of it can be detected, and there is little or no dyspnœa. After a time the pulse falls, and the patient slowly recovers. In four of such cases, after an interval varying from two to five years, valvular disease supervened. Consequently, when cases are met with in which the frequency of the pulse is maintained without any discoverable cause, it is a safe precaution to employ local depletion and mercurial friction over the precordial region.

Chronic heart disease without obstruction of the circulation.

The symptoms in these cases may arise from inorganic causes, for the removal of which suitable treatment must be employed, but which it would be out of place here to specify.

When, however, they arise from organic causes, these are usually such as increase or diminish the contractile power of the heart, with or without inducing some trifling derangement of the valvular apparatus.

When there are evidences of hypertrophy of the ventricles, whether their cavities are dilated or not, the choice of an appropriate treatment is often attended with great difficulties. The immediate effect of this change being to throw a powerful stream of blood into the arteries, and thus fill the capillaries with arterial blood, active depletion, on the plan of Valsalva, has been strongly recommended by Laennec, not only for the purpose of removing the congestion, but with the view of reducing the hypertrophy of the heart itself. Dr. Hope has pointed out the injurious tendency of such extensive venesection, from its inducing a state of anemia with its concomitant evils. But if the views in the tenth chapter of this work are correct, there is another very powerful reason against this treatment; for in proportion as the power of the muscular walls is diminished by it, so is the tendency to dilatation and tricuspid regurgitation increased, whereby congestion of the general venous circulation is produced. With these dangers before us, it is impossible to carry the system of depletion to any great extent. We must be content to relieve the tension of the vessels, when it seems considerable, by small bleedings, or by the application of cupping-glasses and leeches to the precordial region. This plan will often succeed more effectually in quieting the action of the heart than more copious bloodletting, which is often followed by strong reaction. At the same time sedatives and rest materially assist; in fact, it is seldom necessary to abstract blood at all, if the patient be kept free from mental or bodily disturbance. The medicine which most powerfully controls the action of the heart is digitalis. It has with great truth been remarked by Dr. Munck,[1] that with this object the tincture is preferable to the infusion, which in its turn acts more powerfully as a diuretic. But this drug is often very uncertain in its effects, and sometimes produces a state from which the patient is with difficulty recovered; whilst at the same time permanent amendment seldom results from its use. Poppy, hyoscyamus, and conium have appeared to me much safer remedies; but local anodyne friction, particularly with the belladonna liniment, or opium ointment, has seemed to afford more relief than any medicine administered internally. By adopting this line of treatment, we relinquish all attempts to diminish the size of the heart; and notwithstanding the extraordinary cases of Laennec, in which he informs us the heart was reduced to a wrinkled state, it is probable that the generality of experienced practitioners will agree that nothing is lost by thus abandoning the hope of a cure so chimerical, and which is obtained, if at all, by means so hazardous in their nature. Persons treated on the plan above suggested often live on year after year in comparative ease and comfort, greatly relieved, though still retaining a large heart.

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[1] Guy's Hospital Reports.

With a view of preventing that cerebral congestion which many consider to be so much promoted by this state of the heart, it is advisable to employ the douche, or at least cold sponging on the head with regularity.

There is another evil in prospect to which attention must be directed. The same inflammatory action which in so many cases precedes hypertrophy, in many also induces valvular disease. As a prophylactory measure, therefore, mild mercurial frictions may be occasionally used over the precordial region. With the same object, and also for the purpose of preventing inflammations, which are apt to occur in a plethoric state of the valvular system, the diet should not be too stimulating, although sufficiently nutritious to sustain the strength of the patient; by adopting which treatment, the left cavities of the heart will not be irritated by the contact of blood formed from materials of too rich a nature.

If now the action of the heart be enfeebled, it matters not whether this arise from attenuation or from a fatty and flabby state of the walls of the ventricles; in either case a tonic and sedative treatment is clearly indicated. This will in most cases be best carried out by a generous diet, combined with mental repose, and such an amount of exercise as shall invigorate the system without inducing fatigue or hurrying the circulation. The sesquichloride of iron with hyoscyamus is a valuable remedy in such cases.

Cases in which pulmonary obstruction is present.

If there exist both hypertrophy and disease of the valves on the left side of the heart, it is important to know whether the structural changes of the valves are of such a nature as to give rise to mitral regurgitation or not; for if such be not the case, then we must be very careful how we venture to control the action of the heart, because, as has been before shown, this increase of force has the effect of keeping up the circulation by counteracting the effects of mitral and aortic obstruction, and of aortic regurgitation.

If, however, mitral regurgitation takes place, we must, if possible, diminish the energy of the action of the heart in the manner suggested above, but still with great caution; because dilatation of the right side is now more than ever to be dreaded, from the engorgement of the right ventricle produced by pulmonary congestion.

Dr. Henderson has remarked that when aortic regurgitation takes place, if the action of the heart be rendered slower by digitalis, the diastole is prolonged, and the effect of regurgitation is thereby increased.

Nature often gives great relief in these cases by promoting a copious secretion from the mucous membrane of the bronchial tubes, and thus indicates the propriety of expectorant medicines. Of these, squill is one of the most efficacious remedies, and is usually combined with camphor, ether, &c. The pulmonary congestion is also much relieved by the occasional application of leeches or the cupping-glass between the shoulders, and by open blisters on the chest.

When in addition to the signs of valvular disease the action of the heart is feeble, of course the tonic treatment must be combined with the means above mentioned.

Cases in which there are symptoms of general obstruction.

These may exist either with or without those of pulmonary obstruction; that is, without obstruction arising from an imperfect state of the valves on the left side of the heart; for we cannot now consider those cases in which pulmonary disease is also present.

When both the pulmonary and general circulations are thus obstructed the case is very formidable, and is generally drawing towards its close. Still cases are occasionally met with in which much temporary relief is obtained, as in Case LXXIV.

But when there are no signs of disease on the left side of the heart, although it may often be impossible to pronounce whether there is structural derangement of the tricuspid valves, or whether the orifice is simply dilated, yet we can discover important guides to treatment, according as chronic disease of the lungs is present, or as the force of the heart's action is increased or diminished.

We shall conduct our treatment according to the rules above laid down; but we have now also to relieve the engorgement of the capillaries, and to remove the serum that has been poured out. This may be accomplished by increasing the secretions of the kidneys, the bowels, or the skin. The former plan is generally much the most successful. In some cases simple diuretics will succeed, such as a combination of squills, nitric ether, acetate or nitrate of potash, spirit of juniper, &c., with drinks made of cream of tartar, broom top, &c. When these fail, digitalis will often succeed. But as its depressing effect on the nervous system is not merely unnecessary, but in a majority of cases is positively injurious, it must be carefully guarded with ammonia, wine, &c., and it is then a very powerful and safe diuretic.[1]

Its effects may be seen on a reference to Cases LIX, LX, LXXII, &c. &c.

Purgatives, though very valuable in certain forms of dropsy, have seldom been attended with good results in my practice when the cause has lain at the heart. Elaterium often reduces the patient so suddenly, that he rallies with difficulty.

In some cases acupuncturation will afford great relief for a time, but it should not be indiscriminately performed in the scrotum or below the knees, lest it be followed by gangrene. Blisters on the thigh will sometimes also produce a beneficial effect, but are not generally so efficacious as acupuncturation.

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[1] ℞ Infusi digitalis (Nov. Pharm.) ad Oss.
Spiritus ætheris nitrici, ℥ss.
Tincturæ scillæ, ʒij.
Tincturæ cardamomi comp. ℥ss.
Potassæ acetatis, ʒiij.
Ammoniæ sesquicarbonatis, ʒss. Misce.
Sumat ℥j bis vel ter indies.

CHAPTER XIV.

CIRCUMSCRIBED PLEURISY.

The general and physical signs which indicate acute sthenic pleurisy are usually so well marked that the disease is seldom overlooked. There are certain cases, however, where the effused fluid is bounded by adhesions between the opposite surfaces of the pleura, in which the diagnosis is often difficult.

Effused fluid becomes thus circumscribed and confined to some one particular spot of the chest, either by adhesions formed during the same inflammatory attack in which the fluid is secreted, or else by such as have resulted from previous attacks of pleurisy.

The following cases exemplify the former class.

Case LXXVII.

Interlobular pleuritis—Pneumonia.

A wine-porter, æt. 61, a patient of the Birmingham General Dispensary, of temperate habits, and who had enjoyed good health, with the exception of having for a year or two felt a pain and oppression at his heart after hard work, was one day seized with a violent shivering which lasted a short time, and recurred in four hours afterwards. He then felt headache and flying pains, which soon settled around the left nipple. He was seen the next day by the visiting surgeon, and on the following day a consultation was held. His bowels were open, he was very thirsty, and had no appetite. His pulse was 130, full and hard, but rather irregular. His respiration was very laboured, 40 in the minute. The middle of the front of the left side of the chest sounded dull. There appeared a swelling over the precordial region. Bass-viol and cooing sounds were heard all over the chest. The action of the heart was strong, and an occasional click was heard with its sounds like the bursting of a bubble. Venesection was employed freely, and mercury was rubbed in over the heart. The next day the dullness of the left side reached up to the clavicle, and no sound could be heard during respiration over the upper half of the side. Over the rest of the chest were bass-viol and cooing rattles. Expectoration, very adhesive, of an uniform blackish-brown colour, appeared, which, in a few more hours, became ferruginous, and shortly afterwards ceased. Mucous rattles were then heard over the whole of the chest, the pulse gradually lost its power, and he sank five days after his first attack of shivering.

Inspection. The cellular tissue of the mediastinum was filled with air-bubbles. The lungs were of a dark slate-colour and did not collapse as usual. The front of the right lung was emphysematous, and it contained several melanotic masses, from the size of a barleycorn to that of a pea. In one spot the pleura over one of these masses was drawn in and puckered. The left lung was united to the side by tough old adhesions. The upper lobe was in a state of gray softening, very friable, and infiltrated with sero-purulent fluid. The lower lobe was in a state of red softening. In different parts of both lobes were semi-calcareous gray masses as large as a horse-bean. The lining membrane of the bronchial tubes, more especially of those on the left side, was soft, and of a dark reddish-purple colour. Between the two lobes of the left lung four ounces of reddish sero-purulent fluid were found, and the sides of the interlobular fissure were lined with yellow lymph. As the two lobes adhered to each other all around the circumference of the lung, the cavity between them which contained this fluid resembled in shape a melon-seed. The heart was hypertrophied. The lining membrane of the left auricle was opaque and thickened, and of a bright red colour, which tint, however, did not extend to the subjacent parts.

The only diagnosis which could be formed at the onset of this attack was that of intense inflammation about the middle of the left side of the chest. The swelling in that spot, joined to the pain and the dullness on percussion, led to the belief that the pericardium was the chief seat of inflammation. The pulse, it is true, was full and hard, but this might have arisen from some hypertrophy of the heart, which, in fact, did exist. The increasing extent of the dullness upwards, however, joined to the expectoration of ferruginous-coloured sputa, soon revealed the existence of pneumonia also. Effusion from the pleura was not even suspected.

A similar case of interlobular effusion is recorded by Andral,[1] not complicated, as in this case, with pneumonia and endocarditis, but only with a moderate amount of bronchitis and a few crude tubercles.

Case LXXVIII.

Circumscribed pleuritic effusion.

A little girl, æt. 3, usually enjoying good health, was found one evening shivering very much. The next day she complained of a pain in the right side, and became very thirsty and feverish.

When seen in consultation with Mr. Russell, she had a short dry cough, and her respiration was hurried and catching. The pulse was frequent, the skin hot, and the tongue loaded. She had then no pain. The whole of the chest sounded clear on percussion, except the lower part of the right side, which was slightly duller than usual. There were cooing sounds here and there on both sides. For some days after this she seemed to improve under the use of mild alterative and diaphoretic medicines. She was seized rather suddenly with dyspnœa, and in two or three days died.

Inspection. The lungs were slightly congested, and the mucous membrane rather livid in places. The lower lobe of the right lung adhered to the chest, except within two inches of the diaphragm. Between its base and the diaphragm there was more than a quart of rose-coloured, semi-fluid lymph, of the consistence of paper-hanger's paste. The other organs were healthy.

The effusion in this case also was unsuspected, the small amount of dullness on percussion of the right side being attributed to the liver. Nor would the diagnosis have been more correct, probably, had the age of the little patient allowed of her accurately describing her sensations, and of patiently bearing a more protracted examination of the chest. In both these cases the fluid was imbedded as it were in the lung, and thereby prevented from reaching the surface of the chest. There was therefore no dullness on percussion, or diminution of pulmonary sound, proportionate to the amount of effusion.

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[1] Clinique Médicale, Brussels Ed. vol. ii, p. 411.

Case LXXIX.

Pleurisy, with effusion becoming circumscribed.

A boy, æt. 13, had felt a pain in his left side for six or seven weeks, during which time he had cough and some shortness of breath. A week since, he caught cold, and the pain in the side became aggravated, as also the cough and dyspnœa. When admitted into the Birmingham General Hospital, the breathing was distressing, the pulse 120, small and quick, the skin hot, the urine scanty, and the cough was troublesome, and attended with some muco-purulent expectoration. He lay on his left side, which measured, under the nipple, one inch more than the right side. There were traces of œdema under the integuments of this side, the intercostal depressions of which were obliterated. Over the whole of the left side the sound on percussion was very dull. The pulmonary sound was intense over the right side, and absent over the left side, except that a trace of blowing respiration was heard near the root of the lung, which seemed very distant. A large blister was applied and dressed with strong mercurial ointment, and diuretic medicines were administered. For about four weeks he improved, and the fever subsided, but the pulse remained frequent. The respiratory sound was heard as low as the third rib on the left side, and it was also heard at the lower part of the back of this side, but not over the middle. He was then seized with most distressing dyspnœa, and it was determined that if he did not improve in a few hours, paracentesis should be performed. A grooved needle was passed between the fifth and sixth ribs, about three inches from the spine, but no fluid escaped.

Another large blister having been put on, and strong mercurial friction having been employed, he began to improve, and in fourteen days' time the size of the side was greatly reduced, and much rubbing and creaking sound was heard behind it. The middle of the left side in front was now very prominent, and dull on percussion, whilst the lower part of it on percussion gave out an unusually hollow sound, which disappeared on causing the patient to drink a considerable quantity of fluid. No respiratory sound was audible below the fourth rib in front, but it could be heard, coarse and rather feeble, all over the back. Mild mercurial and iodine frictions were kept up, and in three months from the time of his entrance, the prominence over the middle of the left side had nearly disappeared; there was still dullness on percussion over this spot, but some pulmonary sound was heard all over the chest, although more feebly over the prominence above mentioned than elsewhere. Under the nipple the left side measured a quarter of an inch less than the right side. He recovered and left the hospital.

When this boy was first seen, the left pleural cavity was completely filled with fluid, and there was no adhesion between its opposite surfaces. It is curious to remark, that during the absorption of the effused fluid, the whole of the posterior part of the lung should have adhered to the side, whilst fluid remained in front high up in the chest. This probably arose from the patient having lain on the affected side, inclined considerably forwards. Although, therefore, the adhesion, which limited the liquid in the latter part of the time he was in the hospital to the mammary region, took place in the same attack as the effusion, yet, unlike the last cases, it did not arise from partial inflammation, and did not precede the effusion.

A great similarity may be observed between this and Case xc, where, after the fluid had been evacuated, the posterior surface of the lung adhered to the chest long before the anterior surface, but in that case the reasons were very obvious. The patient always leaned towards the aperture that had been made in front of the chest, in the hope of more speedily procuring the removal of the fluid, and the consequent healing up of the wound. There was also a communication between the pleural cavity and the inner surface of the pectoralis muscle, under which the pus had burrowed, and thus formed a pouch which must have closed up by adhesion of its sides, before the anterior part of the lung could adhere to the chest.

In the following cases the fluid was probably circumscribed by old adhesions.

Case LXXX.

Circumscribed pleuritic effusion on the left side.

A schoolboy, æt. 9, ten weeks since, awoke with headache, after which he vomited a green fluid. Two days after this, something seemed to move up and down in the left hypochondrium, which "settled into a pain shooting up the left side of the chest," and, for six or seven hours on one particular day, was very violent. A cough then appeared, which was at first dry, but latterly has been accompanied by the expectoration of a thin clear mucus. On coughing, or walking quickly, he felt a pain in the right side.

When admitted into the Birmingham General Hospital, he was thin and pale; his abdomen was tumid, and his pulse 108, and weak. He was much troubled with dyspnœa on attempting to lie on his right side. He had night perspirations. On a level with the nipple, the left side of the chest measured one inch less than the right side. The whole of the front of the left side, and a portion of that of the right side, between the nipple and the sternum, sounded dull on percussion. The posterior part of the same side was also very slightly duller than that of the right side. The pulmonary sound was intense on the right side of the chest, absent over the whole front of the left side, and feeble but distinct over its posterior part. The heart was seen beating between the right nipple and the sternum, where its natural sounds were heard louder than to the left of the sternum. In three weeks' time some faint and hollow pulmonary sound could be heard as low as the left third rib, and the dullness over that space had almost disappeared. The left side measured an inch and a half less than the right side under the nipple, and rather more than half an inch less under the armpits. The dyspnœa and cough were still troublesome. No further notes of this case exist, but it is known that the boy recovered his health.

There is every reason to think that this lad had previously been attacked by pleurisy on the left side, which had produced adhesion of the back of the lung to the chest, and had caused the ribs to fall in and diminish the size of this side of the thorax. On pleurisy again attacking him, its effusion became necessarily circumscribed by the old adhesions.

Case LXXXI.

Circumscribed pleurisy—Acute pneumonia.

A miller, æt. 22, an habitual spirit-drinker, and subject to severe attacks of articular rheumatism, was attended by the visiting surgeon of the Birmingham Dispensary in one of these attacks, which was accompanied by severe pain in the left side, cough, and expectoration of bloody and ferruginous sputa. He was bled several times, with relief. In seven days the pain and swelling of the joints suddenly disappeared, and the chest symptoms became greatly aggravated. A consultation was held. Respiration took place thirty times in the minute. The pulse was 100, hard. The sputa were very tenacious, but uncoloured. The heart was seen and felt to beat two inches to the right of the sternum under the fourth rib, and its sounds were here heard at their maximum. The lower two thirds of the left side of the thorax were dull on percussion, as also was a portion of it, two inches to the right of the middle of the sternum. Over the upper third of the left side of the chest the pulmonary sound was pure, but intense; in the lower two thirds it was absent, with the exception of a blowing tubal sound heard up the side of the spine. It was natural over the right side, except over the lower portion, where it was rather coarse. Nasal resonance of the voice was strongly marked behind the left side, and slightly behind the right side. A very fine, to-and-fro, rubbing sound, much like the crepitant rattle, was heard over a very small spot, to the right of the lower end of the sternum, which ceased when the respiration was suspended. The complaint progressed, and he died six days from the consultation, and three weeks after his first seizure.

Inspection. The heart was found lying under the sternum, and under the third, fourth, and fifth right costal cartilages.

Immediately to the right of the lower part of the sternum, a rough patch of lymph adhered to the costal pleura over a space as large as a half-crown piece, and a similar patch existed on the pulmonary pleura corresponding to it. In one small spot at its lower and posterior part the right lung was connected to the side by a yellow gelatinous substance. The left lung adhered closely to the thorax in its whole length, from the spine to a distance of four inches outwards, to the whole circumference of its upper third, and to the diaphragm. The left pleural sac contained a pint of serum, in which were numerous masses of soft yellow lymph. It was reduced to a very small size by the adhesion of the lung, and was in the shape of a cone, the base of which corresponded to the middle part of the left lateral region of the chest, and the point was directed on the left auricle of the heart. On inflation, air penetrated and expanded the upper two thirds of the left lung, but only parts of its lower third. This last portion felt more flaccid than it usually does in pneumonia, and contained large patches of a yellowish gray colour, which presented a granulated surface on incision; some little serum, but no pus escaped. These patches were very friable, they did not break down into a pulp, but into a mass more solid than fluid. The back part of the right lung was congested, but crepitated on pressure.

The displacement of the heart to the right side in this case proved the existence of effusion in the left side; and the clearness of the upper third of that side, indicated that the lower two thirds were alone occupied by fluid. If the fluid were free it could hardly have produced this effect, and therefore it was suspected that it was bounded by old adhesions.

Case LXXXII.

Circumscribed chronic pleurisy—Acute phthisis.

A farmer, æt. 23, had enjoyed good health until three months ago, when he had diarrhœa, and pain and swelling in the abdomen, from which he recovered. A week since he awoke in the night with a severe pain in the right side of his chest, which lasted two or three days, and left considerable dyspnœa, with "stitches on deep breathing," but no cough. His pulse was small, 120.

The right side of the chest sounded dull on percussion over its lower two thirds, the level of the dullness varying with position. The pulmonary sound was absent over the same space. There was slight nasal resonance of voice near the angle of the scapula on the right side. A blister was applied to the right side, and dressed with mild mercurial ointment, and diuretic medicines were administered.

In three weeks' time he entered the Birmingham General Hospital. Only the lower half of the right side was then dull, but there was some dullness at the bottom of the left side. The respiratory sound was coarse, and there was dullness on percussion, with fluctuation perceived in the iliac regions, and deep-seated pain on pressure in the left hypochondrium. A creaking sound soon appeared in the right side of the chest. He varied occasionally, but on the whole he emaciated, hectic fever came on, and he sank, the urine never having coagulated by heat or nitric acid.

Inspection. A few ounces of serum were found in each pleural cavity. The right lung adhered to the walls of the thorax by numerous tough white bands, all of them rather less than half an inch in length. Both lungs were slightly congested, but crepitated, and were studded with numerous unsoftened tubercles. The pericardium contained two ounces of serum. The abdominal viscera were glued to each other, and to the omentum. The kidneys were large, and contained a few tubercles.

The line below which dullness on percussing the chest was perceived, varying with the position of the patient, proved the existence of effusion; but, as this extended two thirds up the side, it was naturally concluded that a very large quantity of fluid was effused. It was curious to observe, how a few ounces of serum alone produced this dullness on percussion, by being forced into contact with the surface of the chest, owing to the lung being prevented from collapsing by the adhesive bands, which were just long enough to allow a uniform small space between the lung and the chest, which was occupied by the fluid. It is possible that the effusion was not diminished in the course of the complaint, but that the bands becoming elongated, had increased the distance of the lung from the side, and thereby enlarged the space for the fluid, and lowered its level.

While the foregoing cases prove that, when effusion in the pleural cavity is bounded by adhesion, it will often be very difficult to discover the precise character of the affection, they also show that not unfrequently the diagnosis may be satisfactorily made out, and a careful perusal of them may possibly aid in removing some of the difficulties with which it is surrounded.

CHAPTER XV.

CHRONIC PLEURISY.

It has been remarked with great truth by Dr. Hope,[1] that the "symptoms of chronic pleurisy, with effusion more or less filling one side of the chest, are perfectly well described by systematic writers, yet there is no class of affections more habitually overlooked by the bulk of the profession than this." This has arisen from the general signs which accompany this disease having in some cases been very slight at the onset, and in others almost identical with those of phthisis pulmonalis.

The following cases illustrate the former of these classes.

Case LXXXIII.

Chronic pleurisy, with effusion.

A gentleman of abstemious habits had usually enjoyed good health. After sitting in wet clothes, he lost his appetite, and felt fatigue on slight exertion. This having continued two or three weeks, he was with difficulty persuaded to see a medical man. He complained of no pain, nor of any thoracic symptom whatever; but one day his medical attendant hearing him cough slightly, proceeded to examine his chest, when he found the whole of the left side as dull as possible on percussion; and with this were joined all the physical signs of effusion into the left pleural cavity. Mercurial, purgative, and diuretic medicines were administered. In ten days' time he was extremely feeble, and the fluid remained unabsorbed.

On being sent for to see him, in consultation with Dr. Thompson and Mr. Pritchard, of Stratford-on-Avon, I found that two hours before my arrival, he had nearly fainted, and appeared to be dying, but that he had rallied after the free administration of wine. When visited his countenance was calm, but he could not move in the slightest degree without cough and a sense of suffocation, in consequence of which it was impossible to raise him up. The whole of the anterior part of the left side of the chest was found to be as dull as possible. There was no trace of pulmonary sound over it, nor any vibration during the act of speaking. The heart was displaced to the right side, and beat very faintly, the systolic sound being scarcely perceptible. The pulse was 130, very small and feeble. There was a slight amount of clear frothy expectoration. It was determined to give him wine, ammonia, and beef tea, and to employ mild mercurial frictions over the chest after he should have rallied more.

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[1] Posthumous Paper, Med.-Chirurg. Rev.

A week after this another consultation was held. The pulse was then under 100, and had gained much power. The upper third of the left side of the chest sounded clear on percussion, and pulmonary sound was heard over it. The heart lay under the sternum. The dyspnœa was much diminished. He gradually and very slowly recovered, and is now quite well.

It is hardly possible to record a more instructive lesson than that which is afforded by this case. There was not a single general sign which could have led to the suspicion of any disease of the chest, to which the attention of the practitioner was attracted by a mere accidental circumstance, for the patient himself was not aware that he had any cough at all. Many persons would not have examined the chest under these circumstances, and yet all the while one side of it was full of fluid, and in a short time after the nature of the affection had been detected, the life of the patient was in imminent danger.

Case LXXXIV.

Chronic pleurisy.

A servant girl, æt. 15, was observed to be dull and sluggish, and was in consequence brought for advice. Her breathing was evidently short, and the pulse slightly accelerated. She complained of no pain, could lie on either side, stoutly maintained that there was nothing the matter with her, and stated that she had no cough. On closely questioning her, however, she admitted that she had taken a slight cold fourteen days since, and then felt a stitch in her left side, and that for the last ten days her breath had been rather short. On removing her dress, the left shoulder and the humeral end of the left clavicle were seen to be raised, and the intercostal spaces on the left side were obliterated, nor did the left ribs move during respiration. The heart was felt beating near the right nipple. There was no vibration over the left side of the chest during speaking. Under the armpits the left side measured half an inch, and below the nipple one inch more than the right side. The whole of the left side of the thorax sounded as dull as possible. The pulmonary sound was very intense over the whole of the right side, and absent over the left side of the chest. Behind the left side, near the spine, a faint blowing sound was heard, and the voice sounded distant and squeaking near the root of the lungs. A blister was applied to the chest, and diuretic medicines were administered. In ten days dullness disappeared from the upper half of the left side of the chest, and the pulmonary sound was heard over it. The dullness did not all disappear for seven or eight weeks. Eventually there was very little contraction of the left side.

This is a more common case than the one preceding it, and is one of a large class. The effusion is preceded by pain in the side, but it is of so slight a character that no notice is taken of it, and it is often forgotten. Dyspnœa is only observed on exertion, and the cough has nothing about it to excite attention, being either dry or accompanied by slight, frothy, clear expectoration.

The following case illustrates the mode in which chronic pleurisy sometimes simulates phthisis, and in consequence escapes detection.

Case LXXXV.

Chronic pleurisy simulating phthisis.

A dressmaker's apprentice, æt. 15, had been reported by her medical attendant as being in the last stage of phthisis pulmonalis. She had been ill six months, having at the onset of her illness experienced a severe pain in her left side, which after a few days got better, but it was followed by a cough, and her breath had gradually become shorter and shorter.

When visited she was found extremely emaciated and feeble. Her pulse was 140, very small and weak. She had occasional shivering fits, night perspirations, and evening fever. She had a troublesome cough, accompanied by expectoration of muco-purulent fluid which contained no blood. Dyspnœa was most urgent, and she always lay on the left side. This side of the chest measured an inch more than the right under the nipple, and the heart was felt and seen to beat to the right of the sternum.

The whole of the left side sounded as dull as possible. At the back part of it a trace of distant blowing respiratory sound could be heard near the spine, but nowhere else. On the right side the pulmonary sound was intense, and was accompanied by some cooing sound. There was resonance of voice, nasal and distant, behind the left side near the lower angle of the scapula. A blister was placed on a part of the left side and dressed with mercurial ointment, and a mild iodine ointment was rubbed over the remaining part. Cream of tartar drink was given, and quinine and sulphuric acid. In less than a week the dyspnœa and cough were much diminished, the dullness had disappeared from the upper third of the left side, and the pulmonary sound returned over it. In six weeks some dullness remained, but in six months she had quite recovered. She continues well, having a little curvature of the spine from the contraction of the left side. It is now ten years since the attack.

Perhaps no case could be cited in which the general signs more resembled those of phthisis, and in which, at the same time, the physical signs more surely indicated effusion into the pleural cavity as the principal disease. Time alone could determine whether tubercles also existed in the lung, and it has answered this question in the negative.

It may be thought unnecessary to have detailed cases such as these, which are of common occurrence; but it is because they occur so often, joined to the fact that they are so frequently overlooked, that the necessity has arisen for bringing them forward, and thereby enforcing on medical men the importance of constantly and carefully examining the whole of the chest.

The posthumous paper of Dr. Hope, above quoted, is an earnest that, had his life been spared, he would in all probability have written a very valuable treatise on this subject; but as it is, the following facts, elicited by an analysis of seventy-eight cases of chronic pleurisy, occurring under my own observation, may possibly possess some interest.

Seat. The right side was the seat of the disease in twenty cases, the left in fifty-eight.

Extent. In fifty cases one side of the chest was completely filled with fluid, and in twenty-eight from one to two thirds of the side.

Figure. The chest was enlarged by the effused fluid to a much greater extent on a level with the nipples than under the armpits. In forty-five cases the affected side exceeded the opposite side in measurement by three quarters of an inch, and in some instances by an inch and a half in the former situation, and by about half an inch in the latter. In twenty-eight cases there was little or no difference in the two sides; and in five the affected side measured less than the other, in all of which the fluid only reached up one third of the chest, and the attack, after having been more or less acute, was on the decline at the time of examination.

There was *œdema* of the side affected in twenty cases.

The *intercostal depressions* were more or less obliterated in twenty-eight cases, in all of which the fluid extended up to the clavicle. In two other cases this was observed at the lower third of the chest, after the fluid had been much diminished. In these cases a relapse would appear to have taken place, and the increased amount of fluid secreted was unable to rise higher in the chest, in consequence of the upper part of the lung having adhered to the side. The fluid thus became circumscribed.

Position of the heart. In all the thirty-nine cases in which the left side was full of fluid, the heart was pushed to the right of the sternum. Unless, however, the side was nearly full, the position of the heart was not affected. In only two of the cases in which the right side contained fluid, was it clearly perceived that the heart was tilted up towards the left axilla.

The permanent displacement of the heart to the right side of the chest has been noticed by Dr. Stokes as one of the results of pleurisy, and attributed by him to the absorption of fluid effused into the *right* side of the thorax; an opinion, the accuracy of which he was enabled to verify in one instance after death. As so few cases of this kind are on record, the three which follow may prove interesting, more especially as they in some degree confirm Dr. Stokes's opinion.

Case LXXXVI.

Permanent displacement of the heart to the right side.

A girl, when she was 12 years old, had been run over by a cart, the wheel of which passed over her back and upwards across the right shoulder. She was laid up a long time with very severe pain in her right side. Ever since that time she has suffered from cough, with slight frothy expectoration, and considerable dyspnœa on exertion, and she has felt her heart beat on the right side, which it did not before the accident. On admission into the Birmingham General Hospital, whistling respiratory sound was heard at a considerable distance from her. The left side of the chest measured an inch and a half more round than the right side under the nipple. There was but little action of the right ribs. The lower two thirds of the right side of the chest sounded quite dull on percussion, and the upper third was slightly duller than the corresponding part of the left side. The pulmonary sound was pure but intense over the whole of the left side. It was coarse and feeble under the right clavicle, and absent below this in front. Behind it was distant, blowing, and jerking. Nasal resonance of voice was heard near the root of the right lung. There was no impulse in the precordial region in any position of the patient. The heart was felt to beat between the fourth and fifth ribs external to the right nipple. In that place the healthy sounds of the heart were heard at their maximum. They were also heard clearly behind the right side, but nowhere on the left side.

She has gradually improved up to this time, which is eight years since the accident, and five years since she was first seen. The dyspnœa is now less, but is urgent on exertion. The cough troubles her but little. The heart still beats on the right side, but not so far from the sternum as when first seen. The whistling respiratory sound is as loud as ever. The dullness on the right side is less marked. Both sides of the chest now measure the same. Some feeble and coarsish pulmonary sound is heard all over the front of the right side, very faint towards the bottom, but is totally wanting at the lower half of the posterior part.

Case LXXXVII.

Permanent displacement of the heart to the right side of the chest.

A miner, æt. 53, had been troubled with a dry cough for five years, and dyspnœa on exertion. Twelve months since he took cold, when his breathing became more laborious, and his cough more troublesome, and was accompanied by expectoration of froth, and by a dull aching pain under the left nipple. For the last two months he has had spasmodic cough with scanty expectoration, and great dyspnœa. On his admission into the Birmingham General Hospital, there was slight lateral curvature of the spine towards the left. Below the nipple the right side measured three quarters of an inch less than the left side. The heart was seen and felt to beat to the right of the sternum in a line with the nipple. The left side of the chest sounded clear on percussion, and the lower part particularly so. Over the right clavicle, and three inches below its sternal end, the sound was very dull, and a slight dullness was observed at the spot where the heart was seen to beat. The upper third of the right side behind sounded also dull. Whistling respiration was heard at a distance. Very little respiratory sound was perceived on the right side, but under the right clavicle it was tracheal, and in other parts was masked by dry rattles. Behind the left side there was large, uneven, mucous crackling. He left the hospital in a few weeks relieved, his heart continuing to beat to the right of the sternum.

Case LXXXVIII.

Permanent displacement of the heart to the right side of the chest.

A collier, æt. 39, had been subject to dyspnœa and cough, with muco-purulent expectoration, for ten years, which was attended with increased dyspnœa and constriction of the chest, followed by the expectoration of dark liquid blood on taking fresh cold. He had on these occasions night sweats, and had gradually year by year been losing flesh. Four years ago he caught cold, and suffered much from a severe cutting pain under his right nipple, increased by deep inspiration, and shortly afterwards he noticed his heart beating on the right side of the sternum, where it has remained ever since. On his admission into the Birmingham General Hospital, the left side measured half an inch more than the right side on a level with the nipple. His breathing was rather stridulous. The heart was both seen and felt to beat to the right of the sternum a little below the nipple. The left side of the thorax seemed bulged out and the right side flattened; there was slight spinal curvature to the left. The breathing was chiefly abdominal. The whole of the right side sounded duller than the left on percussion, more especially behind and under the clavicle. The pulmonary sound was coarse, and was prolonged during expiration under both clavicles; it was accompanied by some crackling over the left side, and was quite masked by mucous rattles on the right side. Incomplete pectoriloquy was heard under the right clavicle, and strong buzzing bronchophony below the angle of the right scapula. The sounds of the heart were heard clear and distinct on the right side, but very remotely on the left side. He left the hospital relieved.

The evidences of pleurisy on the right side in Cases LXXXVI and LXXXVIII are strong and conclusive, and the time when the heart passed over to the right side is accurately marked. The general signs in Case LXXXVII are more obscure; but there can be little doubt that effusion had once existed in the right pleural cavity. Indeed the result of such effusion was seen in this as in the other cases, in a diminution of the size of the right side of the thorax.

Dr. Stokes seems to think that the transposition of the heart chiefly arises from the rapidity with which the fluid is absorbed, not allowing time for the ribs to fall in. But in all these cases the side was actually contracted, and the cavity of the right side was diminished. It is probable, therefore, that in the earlier stage of the disease, the lung had been more or less bound down to the spine by adhesive bands, and was prevented from expanding into the space left by the absorption of the fluid, which has become filled up by the heart passing over from the left side. When the same thing occurs on the left side, the right lung expands, and often passes over considerably to the left of the median line.

It is curious to observe that stridulous breathing was heard in all these cases, as if the trachea had been compressed. This must have occurred from the aorta having been dragged across it. This effect was not observed, however, in those cases where the heart was pushed over by fluid in the left side. But in the latter instance the heart meets with a fully inflated lung, which buoys it off from the trachea; whilst in the former it lies on a shrivelled lung, the lower part of which is more or less bound down to the spine.

Displacement of stomach. The stomach is sometimes displaced upwards by the disappearance of fluid in the left side. This took place in Case LXXIX, and also in three other cases in the Birmingham General Hospital, one of whom is now under treatment. In this last case the apex of the lung is evidently bound down, for as the fluid has diminished, the stomach has risen up into the chest, whilst the dullness under the clavicle is nearly as marked as ever it was. In the three former cases, sooner or later, the stomach resumed its normal position, or nearly so.

General signs. Decubitus. In forty-six cases the patient lay on the affected side, in four on the opposite side, and in twenty-eight indifferently.

Pain. No pain was felt from first to last in twenty-one cases; in forty-eight it had been felt at the onset, but only amounted to a stitch, and in most instances had been forgotten at the time of examination. In nine it had commenced with severity. In seventy-two no pain was complained of at the time of examination, and in six there was pain which was not severe.

Dyspnœa. The degrees of dyspnœa glided into each other so insensibly, that it would be impossible to tabulate them. Even when its existence was not acknowledged by the patient, the respiration was seen to be hurried, never being less than thirty-two in the minute, and in some cases reaching fifty-six.

Cough and expectoration. There was no cough in twenty-one cases; in thirty-eight it existed, and was accompanied by clear expectoration in most of them, the quantity of which varied considerably; and in nineteen it was muco-purulent.

Fever. There were hectic fever and night perspirations in ten cases.

Pulse. The pulse was generally small, and often rather sharp; never under eighty, in some cases reaching one hundred and sixty in the minute.

Auscultatory signs. In twenty-four cases the line of dullness on percussion varied with the position of the patient. In four, although the dullness extended only up one third of the chest, its line did not thus change with position. In about one half of the fifty cases, in which the dullness was at first general, it changed with position after the fluid had begun to disappear, but in the other half it did not change. From which it may be inferred that in these latter cases the upper parts of the lung, which were left by the fluid and thus allowed to approach the walls of the chest, adhered to them with rapidity.

The pulmonary sound was hardly ever heard in its pure state. When it was absent on one side, from the presence of a large quantity of fluid, it was generally intense over the opposite side. It was usually either intense or coarse over the upper half of one lung when the lower half was compressed; and in some few cases was decidedly bronchial, being coarse and prolonged during expiration. In these latter cases the effect was doubtless produced by the lung being connected with the chest by adhesions, as in the cases recorded by Dr. Stokes.

In nearly all cases some respiratory sound was heard near the root of the lungs. This it is generally supposed is produced by a small quantity of air entering the lung, which Laennec thinks is never so compressed that it cannot expand a little and thus admit some air. This is undoubtedly true; but the cavity of the chest must be enlarged by the action of the ribs or diaphragm, in order that the air may be forced into the lung by atmospheric pressure. In certain cases, however, this sound was heard at the root of the lung, whilst the ribs and diaphragm were immovable. It is probable therefore that in such cases the sound of the air, passing through the bronchus leading to the *opposite* lung, is conducted to the ear by the condensed lung. The probability of this explanation is increased by the nature of the respiratory sound thus heard, which, although much less intense than that which is heard in pneumonia, has the same hollow, blowing character.

When nasal resonance of the voice was heard near the root of the lungs, it was often impossible to distinguish it from that which is occasionally heard in pneumonia. The pure bleating sound (œgophony) was rarely heard, and was very transitory. Nasal resonance was perceived over the whole surface of the chest of an athletic man in the Birmingham General Hospital, which was filled with fluid. This only confirms the opinion of many, that vocal resonance is one of the least valuable of the auscultatory signs. In pleuritic effusion it must in a great measure depend on accidental circumstances independent of the effusion; such as the degree of condensation of the lung, its freedom from œdema, and its contact with the trachea or bronchus of the opposite lung.

In the great majority of cases no vibration during speaking was felt by the hand placed over the seat of effusion; but this was also observed in CASE LXXXIX (Cancer of the lung).

In more than two thirds of the cases in which one side of the chest was full, some variety of the rubbing sound was heard accompanying the disappearance of the fluid. In two cases it could be heard as a coarse, grating sound, not only by the patient himself, but also by the bystanders at some distance from him. On the ear being placed on the chest of one of these persons, a loud cracking was perceived, which quite startled the listener. Sometimes the sound was creaking when first discovered, but generally it commenced with the rubbing character, and afterwards changed. In about one third of the cases in which it was heard, it was sooner or later accompanied by a fine muco-crepitant rattle, either the one being mixed with the other, or else one being heard in one part of the chest, and the other in another part. This rattle was never heard above the middle of the chest, generally much below it. It remained some time after the disappearance of the attrition-sounds.

From this examination of the signs of chronic pleurisy, it follows that there were no general signs characteristic of the disease, and which clearly indicated its presence, and only one, hurried respiration, which was never absent.

It also follows that the physical signs were sufficient in every case to supply this deficiency.

In consequence of the side having sometimes been found filled with fluid and yet undilated, a great difficulty occurred in determining the nature of the following case, which was at first mistaken for one of chronic pleurisy.

Case LXXXIX.

Cancer of the lung.

A married female, æt. 24, had for some weeks felt a sensation of constriction across the chest, and an increasing difficulty of breathing. At one time she had a pain in the right side, which subsequently left her. She had lost her appetite, was thirsty and very weak. On being visited in consultation with Mr. Russell, her pulse was found to be small, 120; her tongue was foul in the middle, and had very red edges. The whole of the right side of the chest sounded dull on percussion; but the dullness was less marked over its lower half than over its summit. Not a trace of respiratory sound, nor any vocal resonance could be detected on the right side, nor was there any vibration on that side during the act of speaking. The sounds of the heart were natural, and on the left side were not heard beyond the precordial space, but were distinctly audible over the whole of the right side. A puffy swelling was observed above the right clavicle. A blister was applied over the right side, and dressed with mercurial ointment. In ten days' time no improvement had taken place. Both sides of the chest measured the same. The respiratory sound had become whistling, and was audible at a distance from the patient. The voice was also very feeble. Dyspnœa had increased, and she had a slight cough, with some scanty cream-coloured expectoration. Soon after this, difficulty of swallowing came on, she wasted away and died.

Inspection. The whole of the upper half of the right lung was transformed into a mass of encephaloid matter, in which every trace of its natural structure was lost. The upper part was of a yellowish-white colour, was as hard as cartilage, and terminated abruptly at the back. In the front, towards the middle, it was softened into a cream-like mass, which was gradually blended with the remaining part of the lung. This latter was of the colour and consistence of the spleen, and contained some masses of red hardened encephaloid matter, and others which were softer, and resembled custard. Every trace of the right bronchus was lost. The trachea was compressed and flattened, just above its bifurcation. The œsophagus was also compressed by the diseased lung, to which both it and the trachea adhered firmly.

At first sight this case appeared to be one of chronic pleurisy; but this view of its nature was soon abandoned; for, on repeated examinations of the chest, the summit sounded more dull on percussion than the lower part of the right side, except just before death. Active treatment also failed to make the least impression on the disease, and the trachea and œsophagus were seen to be gradually compressed. The true nature of the affection was then suspected, but, from its rarity, it was not confidently diagnosticated. It is worthy of observation, that, although the solidified lung was in contact with the trachea, and by its compression produced a whistling sound, no traces of such sound were transmitted to the ear on the surface of the right side of the chest. This probably arose partly from the right bronchus having been obliterated, and partly from the base of the lung having been converted into a semi-fluid mass, a mixed medium ill adapted for the propagation of sound; so that there was no good conducting substance, either for the aerial or solid vibrations originating at the trachea.

There were five cases, which so far resembled the case of cancer of the lung just related, that whilst the dullness extended up to the clavicle, the affected side was not dilated. In four of them, however, in which the left side was the seat of effusion, the heart was pushed over to the right side. In all the five, some blowing sound was heard at the base of the lung.

Hence there was hardly one of the seventy-eight cases, the nature of which could not be recognised by the physical in combination with the general signs. The greatest difficulty lay in those cases in which the side was not full. But of these there were only five, in which the level of the dullness did not vary with position at one or other period of the disease; and in these there were either attrition-sounds, nasal resonance of voice, or both. When nasal resonance of voice alone existed, the history of the case, or some particular sign, served to distinguish it from one of solidification of the lung.

It would be tedious to enter more fully into the differential diagnosis of this affection, which has been so ably handled by Dr. Stokes.

Terminations. Under the belief that chronic pleurisy was a frequent precursor of phthisis pulmonalis, and with a desire to ascertain whether or no such a belief was well founded, great care was taken during some years to trace the results of the cases, the signs of which have been analysed above. In spite of every effort, however, the subjects of ten of them have been lost sight of subsequent to their recovery. The state of health of fifteen others cannot be exactly ascertained, it is only known that they are living. Of the remaining fifty-three, not one has become phthisical. Fourteen are subject to a cough, in five of whom it existed previously to the pleuritic attack. In all it is accompanied by expectoration of tough, gray-coloured mucus.

When the amount of effused fluid had been extensive, more or less contraction of the side remained for a certain time; but when the patients were examined, two years or upwards after the attack, the affected side of the chest had in many cases nearly resumed its normal proportions, as in Case LXXXVI. So that, at the expiration of that time, one side of the chest was less than the other by an inch in only eight cases; it was a trifle less in fourteen cases; and in the remainder there was no difference in the size of the two sides.

In four, slight vocal resonance remained.

In all, the pulmonary sound was pure, but in five it was rather feeble all over the side that had been affected.

To these might be added numerous other cases, in which the results of pleurisy have been detected both during life and after death, and in which the disease ran its course and terminated favorably without any treatment. From these facts it is hardly too much to deduce that, in general, chronic pleurisy is attended with but little danger either immediate or in prospect. Louis[1] is reported to have stated, in discussing a case of Trousseau's, brought before the Academy of Medicine at Paris by Bricheteau, that he had never met with a case of this kind that had terminated fatally, when the disease was in its simple form.

[1] Med.-Chirurg. Review, July, 1846.

CHAPTER XVI.

TREATMENT OF PLEURISY.

ACUTE PLEURISY.

THE employment of venesection in the treatment of acute pleurisy has almost universally prevailed. Some years ago I was in the habit of ordering it to a considerable extent. It certainly proved efficacious in arresting the disease, but it not unfrequently happened that great prostration ensued, and that a very long time intervened before health and strength were fully restored.

Towards the close of 1837, however, a careful observation of the influenza of that year ripened a conviction which had been some time growing in my mind, that the advantages derived from venesection had been in many cases estimated too highly, and its evils underrated. From that time I have generally refrained from employing this remedy in the treatment of acute pleurisy, especially when it has attacked the artisans of this town and neighbourhood.

When the pain has been severe, local depletion with leeches has been resorted to; in the case of a strong adult twenty having usually been at once applied, by which means a second application has been often rendered unnecessary. But the main dependence has been placed on frictions of the affected side with mercurial and opium ointment.[1]

Between each friction the chest has been covered with large linseed-meal poultices, diluent drinks have been given, and aperient or diuretic medicines, singly or in combination, according to the circumstances of each case.

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[1] ℞ Unguenti hydrargyri fortioris ℥j.
Camphoræ . . . ʒss.
Pulvis opii . . ℈j. Misce.

On the subsidence of the acute symptoms, the friction has been discontinued, and the removal of the effused fluid has been promoted, if necessary, by blisters, and by diuretic medicines.

Under this treatment no case of primary uncomplicated pleurisy terminated in death, however acute the attack. When other organs were affected, or when the pleurisy appeared in the course of some chronic disease, several cases terminated fatally. Thus in Cases LXXVII and LXXXI it was joined with pneumonia; in Case C with pneumonia and tubercles; in LXXVIII with acute bronchitis. In one instance the recovery was apparently complete, but the respiratory murmur remained feeble, and in two years' time tubercles developed themselves in the lung of the affected side. Another case progressed favorably up to a certain point, but some cough and dyspnœa remained behind, and in a few months the same result followed as in the last-mentioned case.

CHRONIC PLEURISY.

The treatment which was adopted in chronic pleurisy depended on the stage of the disease and the state of the patient; whether the case resembled Case LXXXIV or Case LXXXV.

In a great majority of the former cases only mild iodine and mercurial frictions were employed, with opium if any pain existed; and cream of tartar was freely given in the form of imperial. This failing, blisters were applied, and the surface dressed with mild mercurial ointment, and stronger diuretic medicines were administered. In all cases some light nutritious food was ordered, and mineral tonics were speedily resorted to.

In the more advanced cases, large blisters were applied at the onset, and strong mercurial frictions were employed; and it was generally found advisable to support the patient at the same time with wine, quinine, or ammonia, and meat tea.

When fever seemed to exist, it was never increased by these means, but was rather diminished.

Paracentesis thoracis was only performed in one case, or rather an abscess under the skin, caused by the escape of matter from the pleural cavity, was opened.

Case XC.

Empyema—Paracentesis—Recovery.

A young lady, æt. 11, of sanguineous temperament, whilst in the enjoyment of good health was seized with headache, which lasted three days, and was succeeded by a pain in the left side, which was relieved in two days, but soon afterwards reappeared with great severity, and was attended with considerable difficulty of breathing. The day after this she was bled, the violence of the pain subsided, and the breathing became easier in a few days. A cough, however, remained, at first only accompanied by clear frothy expectoration, which latterly had become muco-purulent. In five weeks after her first attack, hectic fever appeared, rigors were succeeded by burning fever and flushes, and night perspirations, with great thirst. She had lain only on the left side all the time, and had become greatly emaciated and extremely feeble. She was brought over in a bed-carriage from Warwick, eight weeks after her first seizure. When visited, she was found extremely emaciated, and so weak that she could not feed herself. She lay on her left side, and the dyspnœa was so urgent that she could with difficulty be raised for examination. The pulse was thready, 130. She complained of no pain, but was teased with a constant cough, with scanty expectoration. She had hectic fever. The ribs of the left side were seen to be immovable, and the intercostal depressions were obliterated. A little below the left nipple there was considerable bulging, which extended from the sternum to the outer edge of the chest, over which fluctuation was very plainly felt. The heart was seen and felt to beat, a little to the right of the sternum. The whole of the left side of the chest sounded dull, very much so in front, and rather less so at the upper part behind. No respiratory sound could be heard over the front of the left side, and only faint traces of it behind. The voice did not cause the left side to vibrate. It was at once determined to cut into the prominence under the left nipple, and the surgeon of the relatives with whom the young lady was staying was summoned.

A free incision was made with a scalpel, and five saucers of very thick, dirty yellow pus, having a most offensive odour, were gradually withdrawn. The next day more or less respiratory sound was heard all down the back, but not in front, of the chest. On striking the front of the left side, about two inches below the outer part of the clavicle, a loud flapping sound was heard, and pressing this part with the finger, a large crackling was felt and heard at a distance, which disappeared after the chest had been some time pressed, when some bubbles of air escaped through the aperture. The cough had almost subsided. The day after this a cupping-glass, fitted with an exhausting syringe, was applied over the opening, and the air gradually withdrawn; after which much pus was brought away, and the respiratory sound became clearly audible under the sternal end of the clavicle. This was repeated two or three times a week, for some time.

Soon after this a compress was applied over the left pectoralis muscle, and the chest was bandaged. To-and-fro rubbing sounds followed by creaking, and traces of fine muco-crepitant rattle, made their appearance in different parts of the left side, and continued for some time. Quinine, wine, and nutritious food were gradually but freely administered, and in sixteen weeks the discharge ceased, and the wound healed up. The pulmonary sound was then heard all over the chest, even close to the edges of the wound. There was, however, considerable spinal curvature to the right, and under the nipple the left side measured an inch and three quarters less than the right side. This difference in size has gradually diminished, and is now (four years since the operation was performed) scarcely perceptible, and the patient is plump and healthy.

The success which followed the operation of paracentesis in this case, where the patient was so much reduced, and the absence of any unfavorable results from the entrance of the external air into the cavity of the pleura, confirms the opinion of Dr. T. Davies, Dr. Rowe, Dr. Hughes, and Mr. Cock, that this operation is in general safe and efficacious. It would have been performed in Case LXXIX, had not the case taken a favorable turn.

But the result is not always so successful as it was in Case XC. The operation was performed some years ago on a young girl, the daughter of a medical gentleman at that time resident in Birmingham. The wound, however, never healed up, and the lung never expanded; so that the cavity of one side of the chest was excessively contracted, and thus caused a great degree of spinal curvature. The poor child lived a few years in this wretched state, and then sank. It must, however, on the whole, be admitted, that there is no great danger or pain attending the operation of paracentesis, if it be performed before the patient has been reduced to a very low state, and with proper precautions.

One of the most important objects to be borne in view, is to ensure the expansion of the lung. The best way of attaining this would appear to be, by the employment of an exhausting syringe, whereby the air being removed from outside the lung, the latter expands, under the force of atmospheric pressure, and bursts the bands by which it is tied down.

A perusal, however, of the terminations of chronic pleurisy, would lead to the conclusion that there is seldom a necessity for having recourse to this operation. In the discussion above alluded to, before the Academy of Medicine of Paris, Louis is reported to have stated that he had seen many cases of pleurisy, but none in which paracentesis was indicated.

CHAPTER XVII.

PLASTIC PNEUMONIA.

There are certain conditions of the parenchyma of the lungs which are considered by all pathologists to be the results of inflammation. These are known by the names of *red* and *gray softening,* or *hepatization,* and *induration.*

The softened state is attributed to acute, and the induration to chronic inflammation.

There are also substances called gray granulations, concerning the nature of which there is a difference of opinion.

Although the red and gray softening are allowed without dispute to be the result of acute inflammation, yet various opinions are held concerning the exact method of their formation. Thus Andral and Dr. Stokes consider the solidity of acute pneumonia to arise not from any deposition of lymph in the air-cells and tubes, but merely from an excessive congestion of blood in the vessels. Andral accounts for the red softening presenting in some cases a granulated, and in others a smooth surface, by the difference in the amount of congestion; stating that "when the tumefaction passes a certain point, its effect is to approximate the cells so closely that they become confounded together, and the granulated appearance vanishes entirely."[1] Dr. Williams, on the other hand, considers the hepatization to arise from the deposition of lymph; and supposes that the granulated appearance is produced by its being deposited in the cells alone, and the smooth surface by its being diffused throughout the other tissues constituting the parenchyma of the lungs.

The extreme minuteness and delicacy of these parts offer a great barrier to the discovery of the exact nature of the diseases affecting them by means of anatomical examination.

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[1] Andral, Path. Anat. vol. i, p. 511.

Accidental circumstances, however, may arise and favour the investigation. Thus Reynaud[1] has shown that in some cases, apparently of ordinary acute pneumonia, lymph is deposited in the interior of the air-vesicles. This he was enabled to demonstrate, by finding the lymph in the minute air-tubes, forming cylinders, which were rendered easy to be detached by suppuration, and were thus drawn out, and with them the mouldings of their vesicular terminations. At the same time few persons who have dried and examined specimens of hepatized lung in the manner recommended by Andral, will hesitate to admit that in some cases hepatization arises chiefly from coagulation of blood in the vessels.

As far then as our limited means of investigation enable us to form an opinion, it would appear that in ordinary acute pneumonia there are two forms, one of which is produced by congestion and inflammatory swelling of the vessels, and the other by the deposition of lymph.

Let us now examine how far this view coincides with what is observed of the effects of inflammation on other serous and mucous membranes which are more open to anatomical investigation, such as the pleura, peritoneum, pericardium, and the mucous membrane of the trachea and bronchial tubes, &c.

In their respective cavities are found :

1. Fluids, consisting of fibrinous or serous dropsy, or increased natural secretions, modified by the inflammatory action.
2. Coagulated lymph or fibrin, with little or no fluid.
3. Both fluid and solid products as mucus, or pus with lymph.

They may be denominated the *serous*, *plastic*, and *sero-plastic* forms, terms relating to the appearance rather than to the chemical composition of such products.

Of these, the sero-plastic is produced by the most violent and acute inflammation; the other two, in the pleural cavity more especially, often arising from a low chronic form of inflammation.

In assigning, therefore, to pneumonia two different forms, the serous and the sero-plastic, we satisfy in part the conditions required by analogy; and it may be further considered as not impossible that the appearance of the surface of an incised portion of hepatized lung may arise from this difference in the character of the exudation rather than from the amount of congestion, as supposed by Andral, or the seat of the deposition of lymph, as held by Dr. Williams; being granulated in the sero-plastic, and smooth in the serous form. In one of Reynaud's cases the granulated state was seen to arise from lymph in the vesicles. "On a careful examination, the plastic matter was traced into the terminal vesicles, which, when they lay under the pleura, presented the appearance of round grains. This substance chiefly resembled fibrin, was white, very slightly tinged with yellow, firmish, and elastic; the colour of the terminal vesicles being slightly gray, or sprinkled with black. Between the different terminal branches there were intervals of a cellular aspect, projecting less both at the surface of the lung and of the incised portion, than the gray grains spoken of."

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[1] Mémoires de l'Académie Royale de Médecine, 1834.

MM. Hourmann and De Chambre found the granulations generally larger in the aged than in the young, which they correctly attribute to the size of the air-cells being usually increased in advanced life.

An extensive series of observations can alone settle the point, which is not of any great moment.

Analogy, however, requires a third form, the *plastic*, in which lymph is deposited with little or no fluid, and this may be looked for in two varieties, in an *aggregated* and a *disseminated* form. No such state having been described, it is proposed to deduce its existence from the following cases. It must, however, be borne in mind, that this form, being in general the result of low chronic inflammation, but few cases of it in its earliest stage can occur to those even who have the most ample opportunities of observing disease; and when they do present themselves, it must almost necessarily be either from death having been caused by the great extent of lung invaded, or else from the coexistence of this with some other fatal disease. In the following case death resulted from the disease having attacked both lungs.

Case XCI.

Acute plastic pneumonia.

A draper's assistant, æt. 21, after exposure to cold was taken ill. On the fourth day of his illness he was seen by Mr. William Hiron, and found to be labouring under pneumonia of the lower half of the right lung, as evidenced by the existence of crepitant rattle and dullness on percussion of the corresponding part of the chest, and traces of rusty-coloured expectoration. On the seventh day a consultation was held, and the upper half of the left lung, which was in a healthy state on the previous day, was found dull on percussion, and coarse blowing respiratory sound was heard over it. There was a very little colourless expectoration. In spite of venesection and the administration of antimony in full doses, the disease progressed in both lungs, but no more moist rattles were heard, and he died twelve days after his first seizure.

Inspection. There was a little lymph between the lower and middle lobes of the right lung. The upper lobe of this lung, otherwise healthy, contained two or three small masses of red induration, the middle lobe was in a state of red induration, and contained some few small patches, of a grayish-yellow colour. The whole of the lower lobe was one mass of solidification, being firm, not easily broken down, of a yellowish-gray colour, and containing a very little yellow-coloured serum, but no pus. The upper lobe of the left lung was in a similar state. The front of the lower lobe was tolerably healthy, and its back part congested. No part of the lungs was in a state of red or gray softening, and very little fluid escaped from them on pressure.

The gray induration in this case exactly resembled in appearance that which is allowed to be the result of chronic pneumonia, except that it was rather more tinged with yellow than is generally observed in such cases. But it differed from it in consistence, being less tough and rather more fragile, but still not very easily broken down. Now this state could not result from chronic thickening of any of the tissues of the lungs by a process of hypertrophy, for the history of the case proves the existence of good health previous to this acute attack of pneumonia. We are, therefore, forced to admit that lymph was very speedily formed without much fluid.

Had this patient survived the acute attack, which he probably would have done had only one lung been invaded, the colour might have become less yellow and the consistence tougher. In the red induration the colouring matter of the blood had not disappeared, and here, also, the toughness of the chronic state was wanting. Thus the remarkable scantiness of the expectoration, whilst other symptoms of acute pneumonia were present, was satisfactorily accounted for.

The suddenness with which consolidation of the left lung occurred forcibly reminded me of what often takes place during the course of typhus fever, which has been mentioned by Dr. Stokes and others, and must have been observed by all persons who have witnessed many cases of fever.

Dr. Stokes argues that the suddenness with which solidification thus takes place is a proof that it arises from congestion alone; but here was an equally rapid solidification, and on dissection this dry, gray induration was found, and was evidently produced by the deposit of lymph. When, therefore, it is remembered that these sudden solidifications in typhus often take place without expectoration, and when once formed are very slowly resolved, it may be conjectured that in some cases, at least, they are caused by the deposition of lymph. Nor is this supposition weakened by the examination of lungs affected by typhoid pneumonia; for the appearance of the diseased parts will be found, as a general rule, to present a more highly granulated surface than in sthenic pneumonia; and although this may partly arise from the dark colour of the blood contrasting with the walls of the vesicles, yet it cannot altogether be thus produced.

Now as the appearance of the patches of chronic pneumonia present also the same difference as those of acute pneumonia in respect to the granulation or smoothness of their incised surfaces, it is possible that the causes of these appearances are the same, and that the granulated form arises from deposition of lymph in the vesicles, whilst the smooth surface is produced either by a deposit of lymph in all the tissues, or only in the blood-vessels which ramify in the walls of the air-cells.

Case XCII.

Diffused plastic pneumonia.

A traveller in the beer trade, æt. 39, formerly of intemperate habits, had been ill eight or nine days, when he was seen by Mr. Alfred Baker. He found him suffering from an attack of acute asthenic bronchitis. The chest sounded clear on percussion throughout its whole extent, and the pulmonary sound, although mixed with rattles, was heard all over it. Being extremely feeble, and in a lodging-house, where he could not receive proper attention, he was sent into the Birmingham General Hospital. The systolic sound of the heart was almost inaudible, and the vital power was very low. His expectoration was muco-purulent. The anterior part of the chest sounded clear; but it was not deemed prudent to raise him for the purpose of examining the posterior portion. Wine and ammonia were freely administered, and he rallied a little. In two or three days' time the lower two thirds of the right side of the thorax sounded very dull on percussion, and presented no trace of respiratory sound. Four days after this he was seized with hemoptysis, and sank in a few hours.

Inspection. The whole of the left lung, and the upper lobe of the right lung were congested with dark-coloured blood, but crepitated. The two lower lobes were totally devoid of air, and dense; of a mottled colour, consisting of a dull white gray spotted with small patches of fawn colour, not larger than the section of rape seeds. On cutting into this solid mass but very little serum escaped. The surface was for the most part smooth; but in some places it was granulated. This part of the lung was not very friable, but resisted considerable pressure between the fingers. There was not the slightest appearance of pus in it.

Here also there appears to have been a rapid deposit of dry lymph in the lungs of a person in a typhoid state. The ordinary characters of acute pneumonia were altogether absent.

The following cases illustrate the deposition of lymph in isolated vesicles.

Case XCIII.

Acute plastic vesicular pneumonia.

A miner, æt. 24, was brought to the Birmingham General Hospital in an open cart, a distance of five miles, on a very cold day in May. He was in a collapsed state. Reaction took place in five hours. It was then found that he had been ill fourteen days, but no clear account of his symptoms during that time could be collected. His skin was hot and dry; his bowels were confined, and his urine was high coloured. His face had a livid and bloated appearance. His intellect was perfect. Dyspnœa was considerable, but he had no cough or expectoration. The back part of each lung sounded rather dull on percussion. The pulmonary sound was masked all over the chest, but particularly behind, by loud cooing and bass-viol sounds, with some muco-crepitant rattle here and there. His pulse was very small, and his debility excessive. The next day the dyspnœa was more urgent, and œdema of the extremities appeared. On rising suddenly from bed, as if threatened with suffocation, he expired.

Inspection. There was a small quantity of clear serum in the left pleural sac. The lungs were of a violet colour, and heavier than usual. On being cut into, some frothy colourless serum exuded. The lower part of the right lung was much congested, but was not very friable. Both lungs were studded with small granular bodies, of a dusky white colour, which felt hard to the touch, but which broke down when pressed strongly between the fingers. At the summit of both lungs they were larger, and had an uneven surface, as if smaller ones were clustered together, and they were more gelatinous and transparent than elsewhere. The mucous membrane of the smaller bronchial tubes was of a violet colour, but no solid deposit could be found in any of them. The heart was large, and its cavities were distended with blood. There was not a trace of tubercular matter in any part of the body.

The granulations in this case were much larger than in any form of ordinary pneumonia, and were separated from each other by greater intervals. The lung, although engorged, crepitated, and was not easily broken down. Very little fluid escaped from it on incision. The history of the case showed the attack to have been acute, and the symptoms indicated suffocation from some interference with the functions of the lung, which, according to the physical signs observed, was not caused either by bronchitis or ordinary pneumonia. Instead of becoming more opaque as they increased in size, which is generally the case with tubercles, the granulations became more transparent.

Case XCIV.

Acute plastic vesicular pneumonia.

A policeman, æt. 25, had been losing his strength and appetite for three or four weeks, and had experienced a "dull sickening kind of headache." Four days since he had been rather suddenly seized with rigors, and on going to bed and covering himself with clothes, he became "burning hot," and at length perspired and was relieved. The next day he felt extremely exhausted, had much nausea and headache, and loathed food; his skin became hot and burning, and his breathing oppressed and laborious. When examined the day after his admission into the Birmingham General Hospital, he complained of a "stupid headache" and "confusion," a nauseous taste in his mouth, and difficulty of breathing, amounting at times to a sense of suffocation. His eyes were watery and injected, his face was rather swollen and livid, more particularly his lips. The tip and edges of his tongue were of a bright red colour, and the middle was covered with a thick, yellow, moist fur. His skin was dry and hot. The bowels were confined. The urine was scanty and high coloured, without containing albumen. The pulse was small, but rather hard, 120. Respiration was short, forty in the minute. He had no cough or expectoration.

The chest sounded well on percussion. Small cooing sounds were heard, more especially over the back, and now and then there was a slight trace of muco-crepitant rattle.

He was leeched between the shoulders. A saline mixture was ordered with oxymel of squills, and simple enemata. In three days' time he had sunk into a typhoid state, with a dark, dry tongue, and great lividity of lips. A slight cough without expectoration appeared. The dyspnœa became urgent, more muco-crepitant rattle was heard, and some dullness was perceived on percussion at the lower and posterior part of both sides. He died on the sixth day of his admission, in spite of every effort to support him.

Inspection. The mucous membrane of the stomach and of the lower part of the ileum was slightly injected in small patches. Both lungs were heavier and redder than usual. They gave out some little colourless serum on incision. They were studded all over with very minute granulations, none of which reached the size of a pin's head, and most of which were not a fourth of this size. They were grayish-white and opaque. A number of incisions showed that they were distinct from each other. They felt hard to the touch, but could be rubbed down between the fingers, although with some difficulty. No deposit could be detected in any of the bronchial tubes, however fine, which were carefully traced with bristles. When a portion of the lung had been soaked three days in water, and another in a solution of alum and nitre, the colour of the whole became paler, and the granulations were less distinctly seen.

The appearance of the lung in this case more nearly resembled ordinary pneumonic hepatization than that described in the last case; but still differed materially from it. The lungs were pervious to air, but more friable than in such cases; whilst the granulations were much larger and less thickly clustered than in any case of pneumonia, whether sthenic or typhoid. The symptoms indicated the march of simple typhoid fever, accompanied by a degree of obstruction at the lungs not sufficiently accounted for by any physical signs.

A case of the same kind occurred in the practice of another physician of the Birmingham General Hospital, in which the appearances of the lung exactly corresponded with those here observed, and in which the symptoms during lifetime were said to have been similar. It should be mentioned that there was no trace of disease in any other part of the bodies, in these cases.

In the following cases the disease ran a more chronic course.

Case XCV.

Chronic plastic vesicular pneumonia.

A student at Cambridge, æt. 21, of high intellectual powers, accustomed to close study, had, during two years, occasionally felt low and sinking, but was always restored by change of air. At length he discovered that his breath was becoming shorter than usual, and he occasionally felt feverish. This persisting for some weeks, he was visited. He was in his usual flesh, and his spirits and appetite were good; his respiration was accelerated, 44, and his pulse was 120, but was not full or hard. The skin was rather hot and dry. The most careful examination of the chest failed to discover anything unnatural in the sounds of respiration, the voice, or the heart. There was no cough, and no expectoration. The dyspnœa continued to increase, and his lips became purple, but still no cough or expectoration appeared, and a consultation was held with Sir Henry Halford.

In another week traces of cooing sounds could be heard over different parts of the chest, and, when gasping for breath, an occasional dry convulsive cough took place. He died in five weeks after he was visited, complaining of no pain, but suffering from the most intense dyspnœa.

Inspection. The lungs were heavier and redder than usual, but crepitated, and were dry on incision. They were studded with gray granulations, the smallest of which were rather less than a pin's head, and were soft and reddish; and which gradually increased in size to that of a rape seed; some of them being semi-transparent and hard. There was no trace of tubercle in any part of the body.

Case XCVI.

Chronic plastic vesicular pneumonia.

A gilt toy-maker, æt. 11, a patient of the Birmingham Dispensary, had been ailing five months, and for the last four or five weeks had been much in the same state as when he was visited. His face and lips were livid, and his body was emaciated.

His respiration was hurried and wheezing, and there was severe dyspnœa. The bowels were regular; the tongue was foul; and the pulse sharp and small, 120. The expectoration was clear and scanty.

The lower third of the left side was dull on percussion. A loud to-and-fro scraping sound was heard, occasionally mixed with traces of fine dry crepitation, over the greater part of the chest, but it was loudest over the lower third of the left side. On the right side some cooing sound existed. The sounds of the heart were natural but feeble. In a week's time no trace of crepitation remained, and the scraping sound was much weaker. The extent of dullness at the lower part of the left side diminished, but the dyspnœa became more and more oppressive. He had no pain, and, in a fortnight after he was visited, he died from gradual suffocation.

Inspection. The pericardium contained three ounces of clear fluid. The heart was healthy. Both lungs adhered to the walls of the chest. On the left side were both old and recent bands of adhesion, and the pleural cavity contained a pint of turbid fluid, in which floated flakes of soft lymph. Both lungs were thickly studded with small round bodies, varying in size from a pin's head to a mustard seed. The smallest were soft, and of pinkish colour; the others were whitish-gray; and some were semi-transparent, and as hard as cartilage. A few of the latter class were grouped together in patches towards the lower half of the left lung; two masses reached the size of a walnut, and presented the appearance of the gray induration so often observed around tubercular cavities. The lining membrane of the smaller bronchial tubes was in places livid, but they contained no lymph. The lungs were heavier and darker than usual; they gave out little fluid on incision, and did not very easily break down under pressure. There was not a tubercle in any part of the body.

In the last case, the granulations were clustered together in some parts, so as to form masses of chronic pneumonia (gray induration).

Other cases of this kind could be adduced, but as some of the granulations were found to be invaded by yellow tuberculous matter, they will be reserved for the chapter on acute phthisis. Combined with tubercles, gray granulations are one of the most frequent lesions of the lungs.

In Cases XCIII and XCIV, the gray matter appeared identical with the typhic deposit of Vogel, consisting of an amorphous, transparent stroma, with some indication of pale cells and granules; but in Case XCVI there were some caudate cells, and even a trace here and there of minute fibres. With the exception of these cells and fibres, the granulations were found to present much the same appearance as that of the gray induration which surrounds tubercular cavities.

In their earliest state, therefore, these bodies approached in their minute structure to the type of typhic and tubercular matter; but seemed to recede further from it in proportion to the time that had elapsed since their formation. This, taken in conjunction with the general history of the cases, would seem to imply that these deposits were formed in a typhoid state of the system; that subsequently the constitution became improved, and that they then showed a tendency to rise in the scale of organization, both from the formation of a morbid blastema having been arrested, and from the increased force of the metamorphosis of tissue, resulting from renewed health.

Supposing the amount of deposit to have been of such moderate extent as not to have interfered with the function of respiration, we may then conceive this process to have been carried still further, and the granulations, whether single or in groups, to have been converted into areolar tissue, and thus to have formed some of the scars and puckerings so often found in the lungs after death.

On the other hand, supposing the typhoid state to have become chronic, it would then closely resemble the tubercular diathesis; in which case the morbid deposit might descend in the scale of organization, and assume the granular appearance of yellow tubercle. Thus would be explained the manner in which fever paves the way for phthisis pulmonalis; the state here described being one intermediate between the two extremes, tubercle and areolar tissue; and having been accidentally revealed by death resulting from other causes, before either of these ulterior changes had taken place.

The connexion, however, of gray granulation with yellow tubercle, will be more fully discussed when the nature of phthisis pulmonalis is considered.

It is submitted, therefore, that as in pleuritis, bronchitis, and other similar inflammations, so in pneumonia there is a *serous, sero-plastic,* and *plastic form;* that the granulated surface of the red and gray softening and induration may in some cases depend on the presence of lymph in the air-cells; and that the semi-transparent gray granulations of the lungs constitute a disseminated form of plastic vesicular pneumonia.

CHAPTER XVIII.

TREATMENT OF PNEUMONIA.

There is a great difficulty in laying down general rules for the treatment of pneumonia, arising out of the opposite forms which it assumes, and the difference in the anatomical character of its several stages. Hence some writers have proposed to vary the treatment with the form, and others with the stage of the disease. But neither of these distinctions can be maintained in practice. For although the extreme cases of the sthenic and typhoid forms require an almost opposite treatment the one from the other, yet, in some respects, they run insensibly into each other. So also, different stages of pneumonia generally coexist in the same lung.

A practical distinction may, however, be drawn between those cases in which pneumonia is the *primary* disease and those in which it is *secondary* to some other affection.

Primary Pneumonia. The employment of bloodletting, mercury, and tartarized antimony, either singly or in combination, constitute the measures hitherto proposed for the treatment of pneumonia. If, however, we desire to form an outline of treatment which, with certain modifications, shall be applicable to all the forms and stages of the disease, bloodletting and mercury must not take the lead. For bloodletting, although very suitable to the sthenic form, is perfectly inadmissible in the asthenic form, unless it consist in the moderate use of leeches or cupping-glasses, as suggested by Dr. Stokes.

On the other hand, although mercury, properly guarded, might not be injurious in the typhoid form, its direct influence in arresting acute inflammations of parenchymatous organs, is more than doubtful, although its value in similar affections of serous and sero-fibrous tissues cannot be questioned.

The influence, however, of tartarized antimony in subduing acute inflammations in general, and pneumonia in particular, has been fully established by Rasori and his followers. It is supposed by many to produce so much depression as to be inadmissible in an adynamic state of the system; but this view will presently be shown to be erroneous. The presumption thus raised in favour of tartarized antimony is further confirmed by the results of Laennec's practice, in whose hands it was employed with the greatest success, and whose accuracy of observation and fidelity of description are well established.

Some years ago, at a season when pneumonia was very prevalent, I had extensive opportunities of witnessing the effects of different modes of treatment in hospital practice, and the results were very much in favour of Laennec's plan. One case was that of a young man who had two attacks of the disease during the previous year, neither of them so severe as that for which he was admitted. In the first attack I saw him treated with repeated venesection and purgatives; in the second, with venesection, calomel, opium, and digitalis. In the third, Laennec's plan was adopted: after one full bleeding six grains of tartarized antimony were administered in the first twenty-four hours, and this was gradually increased to one scruple daily. On the last occasion he recovered three weeks earlier than in either of the previous attacks, and speedily regained his strength.

Having now employed this treatment during the last twelve years, both in hospital and private practice, I can fully confirm the accuracy of Laennec's statement as to its beneficial effects, and venture to assert that, with certain modifications, in individual cases, it is suitable to every form and stage of primary pneumonia.

Of 61 cases thus treated in which the disease was simple, or only complicated with a moderate amount of pleuritis, 3 died, and 58 recovered. One of the fatal cases was that of a man in whom psoriasis of long standing suddenly disappeared under the influence of arsenic, immediately after which a violent attack of pneumonia occurred, for which I was called to attend him. Case xc (double pneumonia) is the second of those who died, and the following is the third.

Case XCVII.

Gangrenous pneumonia—Tubercular matter in the lungs.

A stone-sawyer, æt. 42, six months ago had a fall by which some of his left ribs were fractured, and since that time he has had a cough, which increased three weeks ago, and was accompanied by expectoration of fluid with an extremely fetid odour, and by dyspnœa.

Having become a patient of the Birmingham General Dispensary, his countenance was seen to bear a very anxious cast; his eyes were sunk. The respiration was 44, and the dyspnœa very great. The sputa were of a yellowish-brown colour, rather thin, and very offensive.

There was slight dullness on percussion at the bottom of the posterior part of the left side. Intense pulmonary sound was heard all over the right side of the thorax and over the upper half of the left side. Over the lower half in front it was tubal, and even metallic in spots. Behind the lower half of the left side there was a mixture of fine crepitation and small muco-crepitating rattles. Four days after this he had an attack of hemoptysis, and in ten days more he died.

Inspection. Half a pint of brown-coloured serum was found in the left pleural cavity. The lungs were of a dark slate colour. The left lung was voluminous, and adhered to the side in many points. Immediately under the pleura, as well as in the interior of the lung, were small masses of tubercular matter of cheesy consistence and of a yellowish-brown colour, none of which were so large as a pea. The lower two thirds of the left lung were friable and heavy, engorged with brown serum, which was very offensive, and which imparted to the lung a brown colour. In it were a few patches redder and more solid than the other part. These portions of the lung were insensibly continued into the healthy part, and did not terminate abruptly.

This may be styled a case of gangrenous pneumonia. Although tubercular matter existed in the lungs, yet it had not given rise to any disturbance of the system, and therefore this case cannot be considered as exhibiting the secondary form of the disease.

The ratio of deaths to recoveries was, therefore, much the same as that observed by Laennec.

In Cases LXXVII and LXXXI pneumonia was present, but as it was in both complicated with violent pleurisy and purulent effusion, they have not been included in the sixty-one cases. Indeed they were not treated on the pure antimonial plan, as, in consequence of the extreme violence of the symptoms, venesection was very freely performed and repeated.

When the disease showed itself in its pure sthenic form, one copious bloodletting was practised at the onset, and was immediately followed up by grain doses of antimony. It is very rare, however, to see this form well marked. Both in public and private practice in this neighbourhood it generally from the first assumed a rather lower type, so that in most cases leeches or cupping-glasses were employed, and in many no blood was taken,

The antimony was generally combined with some mild sedative, such as syrup of poppies, and each dose was given in from two to three ounces of fluid, as recommended by Laennec. When these precautions were neglected, it was often difficult to prevent vomiting. When it purged, morphia, either by the mouth, or combined with starch as an injection, usually controlled its action on the bowels. In spite of this precaution, it was necessary to suspend the antimony in six cases, in consequence of its not being tolerated. But in all these cases it seemed to have arrested the progress of the disease. The following case illustrates this.

CASE XCVIII.

Pneumonia.

A schoolboy, æt.6, had an inflammation of the chest at 1½ years of age, and again in his fourth year, and then the hooping-cough. After this he enjoyed good health until a month ago, when he had a slight cough. A fortnight since his cough became worse, and his expectoration was stated by his mother to have been brown and bloody. He had medicine and a blister without relief. When visited as a patient of the Birmingham General Dispensary he complained of a dull pain at the bottom of his right side. The expectoration was ferruginous. There was dullness on percussion, and absence of pulmonary sound at the lower part of the right side behind, and a little above this a crepitant rattle was heard. Much dyspnœa existed, and he could not lie on the right side. The pulse was weak. Four leeches were applied, and half a grain of tartarized antimony was administered every other hour. The next day the breathing was relieved, and the respiration less frequent. The crepitant rattle persisted. The dose of antimony was raised to one grain. In four days he was much better, the sputa being still brownish. The antimony not being tolerated, was omitted. In another week no crepitant rattle could be heard; the respiration was natural, and he could lie on his right side. All medicine was discontinued. In two or three days more he was quite well.

Many of these cases were complicated with slight pleurisy, which was generally accompanied by effusion. A few leeches usually removed all pain from this cause; and when this remedy failed, mild mercurial ointment with opium was rubbed in, and in some instances a blister was applied over the seat of pain and was dressed with this ointment.

Dr. Alison has mentioned a sensation of heat in the chest as often preceding an attack of pneumonia. This is seldom observed by the physician either in hospital or private practice, on account of his rarely seeing the patient at so early a stage of his malady. Not unfrequently, however, this sensation is felt throughout a great part of his illness. When this occurred, or when the skin felt very hot, cold wet cloths were applied over the chest with great relief to the patient.

When the vital powers were depressed at the onset, antimony was still given; but with it were ordered ammonia, quinine, wine, beef tea, &c., according to circumstances. With one or two exceptions, the remedy was well borne when the system was thus supported.

Case XCIX.

Typhoid pneumonia.

A married woman, æt. 33, was affected three weeks since with cold in the head and coryza, followed by cough with yellow thick expectoration. She took to her bed, and suffered from headaches, and her tongue became dry and black. She was delirious. When admitted into the Birmingham General Hospital she expectorated a small quantity of clear and very adhesive mucus. The lips were dark and sore, and the tongue was dry and covered with dark sordes. The pulse was very small and frequent, but rather sharp. The lower two thirds of the posterior part of the thorax sounded dull on percussion. Over this part a strongly marked blowing sound was heard, and nasal resonance of voice. The systolic sound of the heart resembled the diastolic sound in quality, and was very faint. A grain of tartarized antimony was ordered every four hours, and between every dose two grains of quinine, and this was continued for two days, when the dose of antimony was increased to two grains. The pulse was then stronger, larger, and softer. A good deal of muco-crepitating rattle was heard at the middle of the right side of the back. The dullness remained. In three days' time the strength of the patient was much increased; the rattle was spreading downwards, but the appetite was very bad. The antimony was omitted, and a blister was applied to the chest. In two days the appetite improved. In a week or two she completely recovered.

In most cases the antimony was continued for some time after the urgent symptoms had disappeared, as recommended by Dr. Graves and Dr. Stokes. Having been discontinued on one or two occasions, a relapse took place. The following case affords an instance of this.

CASE C.

Typhoid pneumonia—Relapse.

A glass-cutter, æt. 51, had a slight cough ten days ago, in three days afterwards he had a shivering fit, since which time his breath has been getting very short, and he has felt a great sensation of tightness across his chest, and has lost his appetite. When visited as a patient of the Birmingham Dispensary his countenance was pale and haggard, and his eyes sunken. His pulse was small and feeble, 104; his skin was dry, but not hot. He could lie best on his left side. The expectoration was sepia coloured throughout, and adhered to the sides of the vessel.

There was complete dullness over the lower two thirds of the left side of the chest. Over the same space a bronchial, blowing sound was very distinct, exactly as if some person was breathing into the ear of the listener. The pulmonary sound under the clavicles and over the right side was intense. There was no trace of any moist rattles. Pectoriloquy was heard over the lower two thirds of the left side of the chest, perfect below the angle of the scapula, and doubtful in the other parts. The respiration was laboured, 40. One grain of tartarized antimony was ordered every other hour. The dose was gradually raised to gr. iij, and persevered in for four days; at the end of that time the sputa resembled that of common catarrh; a little crepitant rattle was heard behind the left side; bronchophony replaced pectoriloquy; but the blowing sound, though less marked, still remained. The antimony was omitted, as there existed hiccup with great prostration of strength, and because he had never tolerated it well, being much purged when he did not vomit. One minim of Scheele's hydrocyanic acid was administered every four hours, which soon removed the hiccup, and effervescing mixture was given with beef tea. He gained strength and improved in every respect for ten days, when he was suddenly seized with a relapse. The sputa became ferruginous, the crepitant rattle disappeared, except just at the base of the lung, and his strength fell rapidly. His appearance was ghastly, and his pulse was small and extremely feeble. Antimony was again administered, but after four days he could bear it no longer. A great improvement, however, took place in a day or two; the sputa became colourless, but the dullness, bronchophony, and blowing sound were a long time in disappearing, and the crepitant rattle, which had returned, seemed to become moister, larger, and more uneven at the base of the lungs, thus being changed into the muco-crepitant rattle. The patient eventually recovered, and in three months had regained his flesh.

This man would in all probability have done better had he been more supported from the first; but at that time experience had not taught me the necessity for this, particularly among the artisans of the town, nor had I learnt to appreciate the value of the combination of tonic and sedative remedies.

This case occurred in 1837, when the influenza raged with much violence; and in four other cases of primary pneumonia which came under notice at this time, the expectoration had the same sepia colour, and they were all of a very adynamic character.

Thus in all these cases the administration of antimony formed the leading feature in the treatment, being combined with venesection or tonics, with their intermediate grades, according to the state of the individual. In four of them the third stage had commenced in some portion of the lung prior to their coming under treatment, at least if prune-juice sputa be considered as its characteristic sign.

It may be thought unnecessary to have thus advocated Laennec's plan of treatment, as many practitioners of great experience, amongst whom are Louis and Andral, have pronounced in its favour, although they at one time doubted its efficacy. Few persons, however, have employed it in the typhoid form in combination with tonic remedies, as here suggested. There is, indeed, sometimes great indecision exhibited in the treatment of pneumonia, and a frequent change of remedies which is injurious to the patient; and when antimony is given, it is too often administered in small doses, when, although it may nauseate, it has little or no effect in arresting acute attacks of the disease. It is possible, therefore, that some may be encouraged to pursue a more decided line of practice by the perusal of these pages.

SECONDARY PNEUMONIA. When pneumonia occurs in the course of other affections, or after injuries or operations, it not merely assumes the typhoid, but also the latent form, in the great majority of cases; more particularly as seen in hospital practice. And as in these cases solidification often takes place with great rapidity, it not unfrequently happens that the disease has existed for some time before it is discovered.

Hence the importance of frequently examining the chest under such circumstances. Were this done oftener, the mortality which occurs after injuries and operations would probably be much diminished.

The following case illustrates the insidious manner in which the disease advances under these circumstances.

Case CI.

Latent pneumonia after injury to the knee.

A gentleman, æt. 43, of leuco-phlegmatic temperament and rather stout, was riding, when his horse fell with him and injured his knee. Suppuration took place, and several incisions were rendered necessary. This reduced him very much, but he was slowly recovering, when he took cold, as he thought, by sitting at an open window. Slight dyspnœa and cough appeared, without expectoration, but his leading symptoms were debility and loss of appetite. Seen in consultation with Mr. Wood, the action of the heart was feeble, and the pulse small and frequent. There was great prostration, and nervous excitement, with an anxious expression of countenance. The tongue was rather dry and coated. Respiration 40.

On percussing the thorax the lower half of the right side was dull, particularly at the posterior part; and over this portion of the chest there was a faint, blowing respiratory sound, and some traces of nasal resonance. In other parts a slight cooing sound might be occasionally heard. The sounds of the heart were weak. Ammonia and ether, with tincture of squills, were administered, and a blister was placed on the right side and dressed with mercurial ointment. The general symptoms were relieved in a few days, but the dullness and absence of healthy respiratory sound continued a long time, and when this latter reappeared it was very gradually, and was unaccompanied by any moist rattle whatever.

Now, as in this class of cases the system has been previously very much reduced, and as a fresh shock might be fatal, I have never ventured on the administration of antimony, lest violent vomiting or purging should be thereby induced, and be attended with fatal results. Otherwise could we be certain that it would be speedily tolerated, the same treatment might be adopted as in the primary asthenic form.

Mercury, opium, and counter-irritation, with a considerable quantity of quinine, ammonia, and wine, according to the circumstance of each case, have been usually employed.

Of course no particular treatment can be pursued in those cases in which the plastic form predominates, because we can never say when such is the case.

CHAPTER XIX.

NATURE, DEVELOPMENT, AND CAUSES OF PHTHISIS PULMONALIS.

NATURE.

The term phthisis pulmonalis is here employed to denote that form of disease in which the lungs are found to contain masses "or tumours of yellowish white colour, of dull aspect, free from glossiness, and of variable consistence,"[1] called tubercular matter.

The most successful and delicate injections have failed to demonstrate the existence of blood-vessels in tubercular matter. Chemical analysis has shown it to consist of protein-compounds, fat, and earthy salts, in variable proportions. Viewed under the microscope it is seen to be composed of three elements.

1. An amorphous, transparent stroma, which reacts chemically like coagulated fibrin.

2. Minute granules, some of which consist of fat, some of protein-compounds, and others of earthy salts.

3. Corpuscles, generally about one third of the size of pus-cells. According to Lebert and Dr. Hughes Bennett, they never contain nuclei, but, according to Vogel and Gulliver, they are often nucleated. It appears to me that Dr. Hughes Bennett has figured as cells, the same corpuscles which Vogel denominates nuclei. It is certain that a faint appearance of a cell-wall may sometimes be seen around these corpuscles; at other times there are traces of such cells without any nuclei. All observers, however, are agreed in considering them as imperfectly developed cells. In recent firm tubercle, the amorphous stroma and cytoblasts predominate; the latter being often closely clustered together. In soft tubercles they are generally farther apart, and less numerous, whilst the granules are greatly increased.

These elements also occur in individual cases in different proportions.

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[1] Syden. Soc. ed. of Louis, p. 2.

Tubercular matter, therefore, contains the elements of nutrition in a degraded form, and may be considered as a product of abnormal nutrition, in which an abortive attempt at organization has taken place.

As matter of a similar appearance, and of the same ultimate structure, is found in various parts of the body, phthisis pulmonalis cannot be esteemed a local disease of the lungs; and as Vogel has further shown that a substance of the same histological character is secreted in some forms of typhoid fever, and in scrofula, phthisis pulmonalis is now universally considered, like these diseases, to depend on a peculiar derangement of the constitution, denominated the tubercular diathesis, or tuberculosis.

In what this derangement consists we know not. Some pathologists state that in the early stages of the disease, the blood contains less fat than it does in health; whilst others have been unable to discover any such alteration. In its more advanced stages, Andral and Gavarret[1] have detected an increase of fibrin in proportion to the number of blood-corpuscles; but the same change is found in other disorders, particularly those of a decidedly inflammatory character. This state of the blood may possibly be accounted for by the acceleration of the circulation which usually takes place soon after the disease has set in; by which means Simon[2] supposes the blood-corpuscles are consumed more quickly in the lungs than they are formed in the chyle, and that hence arises the disproportion which exists between them and the fibrin.

Whether any specific matter exists in the blood in typhus, scrofula, or tuberculosis, cannot be ascertained. If there be such, chemical analysis has hitherto failed to detect it.

Looking, however, to the usual course of phthisis, and to the analogy which exists between it and the above-mentioned disorders, we are led to the conclusion that, like them, the tubercular diathesis is associated with an asthenic state of the system.

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[1] Annales de Chimie et de Physique, vol. lxxv, p. 225.

[2] Simon's Chemistry. Syd. ed. p. 286.

DEVELOPMENT.

Tubercular matter is never seen in any other than the solid form. Vogel,[1] however, thinks "there can be no doubt that its formative substance is secreted from the capillary vessels in a fluid form, and that it afterwards fills up the interstices of the tissues in a manner too perfect to be accomplished by any substance that was not originally fluid." This is admitted by Andral.[2] It may therefore be considered to be formed from an amorphous fluid blastema, like pus and other morbid products, the development of which has been seen to take place in this manner.[3] The supposition of Addison, that tubercle is formed by the accumulation of abnormal epithelial cells, seems quite irreconcilable with the received views of nutrition.

The formation of tubercular matter may be considered to take place in three ways.

1. It may be directly formed from the fluid blastema as a single product. Thus the smallest grains of tubercular matter have, not unfrequently, the same appearance and ultimate structure as the larger masses, being yellow and opaque, and having the granular elements numerous in proportion to the corpuscles.

2. Other products may be simultaneously formed with it from the same blastema. This is supposed by Carswell to take place very frequently. He observes, "This process of separation of tubercle from secreted fluids is strikingly exemplified in tubercular peritonitis. When we examine the peritoneum thus affected, the three following stages of the process are frequently extremely well marked: First, on one portion of this membrane there is seen a quantity of recently secreted coagulable lymph; secondly, on another we find the same plastic semitransparent substance, partly organized, and including within it or surrounding a globular mass of tubercular matter; and lastly, on another part, the coagulable lymph is found converted into vascular or pale cellular tissue, covered by an accidental serous

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[1] Op. cit. p. 277.

[2] Med. Times, No. 384, Feb. 1847, p. 358.

[3] Op. cit. p. 108.

membrane, beneath which, and external to the peritoneal or original secreting surface, the tuberculous matter is seated, having the form of a round granular eminence resembling in colour and consistence pale, firm cheese. In this, as well as in the preceding case, we cannot but perceive that the formation of tuberculous matter originates in a process similar to that of secretion, that its separation from the blood may be accompanied with that of natural and also other morbid secretions; and hence the reason why its physical characters are sometimes obscured, particularly in the first stages of its formation."[1]

3. It may be preceded by the formation of other products of abnormal nutrition. Accordingly a gray semitransparent matter is frequently found associated with and preceding the appearance of tubercles in the lungs, and occurring in three forms.

1. As small roundish tumours called gray granulations. 2. In masses surrounding cavities. 3. In considerable sized masses inclosing a few yellow tubercles.

The first two forms are extremely common. Louis states that he hardly ever met with a case of yellow tubercles without the presence of gray granulations.

The third form is by no means of frequent occurrence. It is, however, of the utmost importance to study its development and its connexion with tubercles, because much light is thereby thrown on the nature of the process which precedes the appearance of the latter bodies, and which under other circumstances is hidden from our view.

The following cases will be found to illustrate the truth of this remark.

Case CII.

Tubercular cavity surrounded by red and gray induration—Pneumonia.

A female servant, æt. 19, had been exposed to cold when in a state of profuse perspiration; the catamenia were suddenly arrested, and had not reappeared for the last four months. When examined as an out-patient of Addenbroke's Hospital, Cambridge, she complained of lassitude and great dyspnœa on exertion. The countenance was pallid, the pulse small and feeble. The bowels were confined. She had a trifling cough, with little or no expectoration. Slight dullness was perceived on percussion under the left clavicle. In this spot there was a trace of vocal resonance, and the pulmonary sound was coarse, and was prolonged during expiration.

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[1] Carswell's Pathology—Tubercle.

Iron and aloes were administered, and a blister was applied to the chest. Three weeks after this she became feverish and much worse, and was then admitted into the hospital.

Immediately under the left clavicle there were decided dullness of sound and a very circumscribed mucous rattle. The lower half of the same side was also dull, a fine crepitation was heard over it, and here and there some bronchophony. The general signs of pneumonia were also present. She improved for two or three days under the use of Laennec's tartar-emetic treatment. She was then seized with a very sharp pain under the left false ribs, chiefly on inspiration, which consequently became jerking. She was compelled to sit up and lean forward. She was bled, and the next day she died.

Inspection. There was a little œdema at the summit of the right lung, which was otherwise healthy. In the apex of the left lung was a tubercular cavity, as large as a hazel nut, surrounded by a mass of red induration, in which were some patches of gray induration and a few unsoftened, opaque yellow tubercles. The lower half of this lung was much engorged, and gave out some bloody serum on pressure. In it were two or three masses of red hepatization, each as large as a walnut. Two ounces of reddish serum were found in the left pleural cavity, and a finely injected patch of lymph existed on the centre of the diaphragmatic portion of the left pleura.

The history of this case leads to the supposition that on the cessation of the catamenia blood was determined to the summit of the left lung, was followed by the secretion of gray matter, and subsequently by the appearance of yellow tubercles. It is not certain, however, that these latter bodies were not first deposited, and those who maintain that they irritate the lung as foreign bodies, would doubtless assume such to have been the case. The same objection will not hold in the following case.

Case CIII.

Tubercular peritonitis—Tubercles in the lungs surrounded by red and gray induration.

A rule-maker, æt. 17, had lost flesh rapidly of late, and had felt pain in the abdomen for eight weeks. On admission into the Birmingham General Hospital he was seen to be very pale and thin. His pulse was small and frequent. He had a slight cough, which, during the last four days only, had been followed by thick yellow expectoration. The urine was scanty, but not albuminous. The abdomen was tumid, having been gradually swelling for some weeks. There was manifest fluctuation in it; and the lower two thirds of it sounded dull on percussion, the level of the dullness varying with the position of the body.

There was some dullness under the left clavicle, in which spot the respiratory sound was prolonged during expiration. There was very slight resonance of voice under both clavicles. Ten days after this a mucous click was heard, with bronchial sound, under the left clavicle, whilst under the right the pulmonary sound was rather coarse, and slightly prolonged during expiration. In another ten days his cough for the first time became very troublesome. Mucous crackling, slight pectoriloquy, and dullness on percussion were perceived under the left clavicle, and under the right, buzzing bronchophony, with very coarse bronchial sound prolonged during expiration.

Twenty-four days after his admission he seemed as well as usual; but the next morning the nurse stated, that he suddenly "altered," complained of some slight increase of pain in his abdomen, and began to vomit. He died about eight hours afterwards.

Inspection. Both pleuræ were milky and opaque, and were studded with flattened patches of cheesy tubercular matter. At the summit of the right lung were some masses of red induration as large as walnuts, and not very hard; their section was granular. In the very apex of the left lung was a cavity, three and a half inches by one and a half, containing some cheesy tubercular matter, part of which was softened. Around and below this cavity were some masses of red induration mixed with gray matter, and also many gray granulations. The abdomen contained a brown liquid, having a fecal odour. The peritoneum was universally thickened and opaque, and was studded with flattened patches of tubercular matter. The intestines were glued to each other and to the peritoneum. Groups of small ulcerations existed above the ilio-cæcal valve. In the centre of two of these were perforations opening into the peritoneal cavity.

Here Nature was surprised at her work. Certain physical signs indicative of the deposition of solid matter, were observed in the summit of the left lung, followed by others denoting tubercular softening. In the mean time, the signs of solidification in the apex of the left lung appeared, when the patient was suddenly cut off by perforation of the small intestine. Tubercular matter was found in the left lung, surrounded by gray and red induration; but in the right lung there was red induration alone, without a trace of gray or yellow matter. This red induration was caused by lymph, in which some of the red corpuscles of the blood remained. In this case, therefore, there was first determination of blood, then a deposition of lymph, and lastly, tubercular matter appeared.

Case CIV.

Gray induration of the lung, inclosing a few tubercles.

A maid-servant, æt. 35, of intemperate habits, who had been much exposed to cold and damp, and who had experienced two or three severe attacks of rheumatism, was admitted into the Birmingham General Hospital, labouring under articular rheumatism. She stated that, for some months, she had suffered from cough and dyspnœa. The greater part of the left side of the chest sounded dull on percussion. An occasional click was heard under the left clavicle, but very little respiratory murmur. No unusual sound was detected in the precordial region. In a few days she died rather suddenly from dyspnœa.

Inspection. Both lungs were congested, but the upper half of the left lung was in a state of gray and slate-coloured induration, and in the midst of it were several small tubercular cavities, and some few yellow tubercles. No trace of tubercle was found in any other part of the body. Some gray granulations in clusters resembling gray induration were seen in the right lung. The back flap of the mitral valve adhered closely to the ventricle; the other flap had thickened edges, and short chordæ tendineæ.

We are forced to admit the same order of sequence in this case also. For we cannot suppose that the few tubercles which existed could have given rise to such an amount of irritation, as to have produced the great mass of gray induration. Besides, similar matter was found in the other lung, without a trace of tubercle near it.

The same appearances were observed in Cases VII and XXIII, in which the induration of the lung was clearly produced by the irritation of an aneurismal tumour. Reynaud[1] relates the case of a lad, æt. 15, the upper half of whose left lung was converted into a mass of yellowish-white colour, tolerably firm, presenting a smooth surface on incision, and a granulated appearance when torn, in the middle of which was a tubercular cavity. On carefully examining this portion of lung, the smaller bronchial tubes were found to be filled with tubercular matter; "but a point which it is important to remark," says Reynaud, "is, that this matter had not a tubercular appearance in the larger tubes, but rather resembled the plastic matter which forms ordinary false membranes." It would appear, therefore, that, as in the preceding cases, the lymph which had been longest secreted was converted into, or replaced by tubercular matter.

It has never been denied that, in the form of granulations, the gray matter precedes the formation of yellow tubercle; and I think it will be allowed to follow, from the above cases, that the same takes place when larger masses of it are found in the lung, surrounding a few tubercles. It is indeed the same process as that which goes on when tubercles are formed in false membranous bands, which must have existed before the tubercles could have been deposited in them, or formed out of them.

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[1] Mémoires de l'Académie Royale de Médecine, vol. xiv, pp. 160, 163.

When only a small portion of gray induration is found surrounding tubercular cavities, it is very probable that in most cases its formation is subsequent to that of the tubercular matter, which gave rise to the cavities.

All pathologists agree in considering the gray masses above described as the result of chronic inflammation. They may, therefore, be viewed as consisting of coagulable lymph, incapable of reaching the degree of organization attained by that which is thrown out in more active inflammation, occurring in a sthenic state of the constitution.

Different opinions, however, are held concerning the nature of the gray granulations. Laennec and Louis consider them to be essentially tubercular, "a stage through which yellow tubercle must necessarily pass." Carswell looks upon them as formed by inspissated mucus in the air-vesicles, which frequently incloses a portion of tubercular matter. Andral looks on them as air-cells thickened by inflammation, and this view is also taken by Dr. Alison, Dr. Williams, and others. Vogel, Lebert, and Dr. Hughes Bennett describe them as composed of the same elements as yellow tubercle; with this difference, according to Vogel, that the amorphous stroma and corpuscles are more numerous in proportion to the granules than in yellow tubercle; to the scarcity of which granules, he supposes, they owe their transparency. But if they are compared with certain forms of lymph, for instance, with gray induration of the lung, with vegetations on the mitral valve, and horny patches on the aorta, it will be found that they approach these latter quite as closely in minute structure as they approach yellow tubercle. It has been shown by Vogel, that tubercular matter cannot be distinguished from typhic deposit and some other morbid epigeneses. In the chapter on Plastic Pneumonia, it was seen that, in certain cases, the minute structure of gray granulations approached towards that of fibrous tissue. If, therefore, gray granulations were classed on the one hand with tubercular matter, on account of their histological character, they might on the same grounds be classed with typhic deposit. Few persons, however, would venture to pronounce yellow tubercle, gray granulation, and typhic deposit to be the same substances, although equally few would deny such an amount of affinity between them in some points, as might lead to the belief that, under certain circumstances, the one might readily be converted into the other.

Considerations, therefore, based on the general appearance of the gray matter, its mode of development in certain cases, and its minute structure, lead me to conclude that in all its forms it is an imperfectly organized substance, which, in the absence of the tubercular diathesis, may remain stationary, or may rise slightly in the scale of organization, but which, under opposite circumstances, may descend in the scale and be converted into or be displaced by tubercular matter. In this sense it may in some cases be the first stage of tubercle, but not a stage, as supposed by Laennec[1] and Louis[2], " through which tubercles must necessarily pass before acquiring their own peculiar character," nor must it of necessity be followed by tubercle.

It is impossible to determine by what process yellow tubercle is developed in the midst of the gray matter; whether it is deposited in it as a nidus, in the same manner as earthy salts are deposited in atheroma, or whether the gray matter is transformed into it as lymph is converted into pus. Both suppositions may be reconciled with our present knowledge. Thus, if we suppose tubercle to be deposited in the gray matter, this process must be the result of an increasing tubercular diathesis, whereby a blastema highly charged with tubercular matter is thrown out. But if the gray matter after remaining a certain time is transformed into tubercle, it must be in consequence of a diminution in the activity of the metamorphosis of tissue, by the force of which it had been hitherto kept from descending in the scale of organization. This brings us to the same point; for the activity of the metamorphosis of tissue must in its turn have been diminished by derangement of the system, which had taken place under the increasing influence of the tubercular diathesis. Both theories, therefore, are consistent with the fact, that while in the process of normal nutrition the products are mainly influenced by the metamorphosis of tissue, in abnormal secretions the quality of the blastema derived from the blood is mainly concerned in modifying the nature of the morbid product.

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[1] Traité d'Auscultation.

[2] Op. cit. p. 2.

It has been proved that normal tissue cannot be directly converted into pathological formations, which, on the contrary, arise out of an amorphous blastema. Gray matter, however, is not normal tissue, but an imperfectly organized substance. Both tubercle and gray matter are protein-compounds, of the transformations of which we are as yet ignorant; so that, to say the least, there is nothing improbable in the notion that the one is transformed into the other.

Engel[1] seems to take this view of the origin of tubercles, considering one kind to be the result of a "peculiar state of the blood, closely approaching in its state to typhus, and the other in all cases being an inflammatory product;" and he thinks that the conversion of inflammatory exudation into tubercular matter, and not into other structures, depends on various conditions connected both with the quality of the exudation and the degree of activity of the metamorphosis of tissue.

No practical advantage would be gained by pursuing this inquiry further.

It follows, from what has been shown as to the mode of development of tubercular matter in the lungs, that it must be preceded by local congestion. Vogel[2] considers it to "result from the same causes as fibrinous dropsy generally, and to be preceded by local hypcremia of the participating capillaries." Andral[3] states that the "pre-existence of congestion may be admitted but cannot be demonstrated." Capillary congestion may be distinctly perceived preceding the deposition of typhic and scrofulous matter in the mesenteric glands. It is more difficult to observe this in the lungs, but in acute phthisis it may be occasionally seen. In chronic phthisis, the results of such congestion alone meet the eye; but it will hardly be denied that the deposition of lymph, although of a low character, proves the pre-existence of some amount of congestion. Besides this, physical signs of congestion are often observed at the summit of the lungs, which in some cases totally disappear, and are in others succeeded by tubercular cavities. The progress of such congestions may be traced in the cases above detailed.

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[1] Day's Vogel, p. 287.

[2] Op. cit. p. 277.

[3] Med. Times, Feb. 1847, p. 358.

Whilst the meaning attached to the word inflammation remains so indefinite as it is at present, it would be useless to discuss whether the action which accompanies the deposition of tubercle is of an inflammatory nature or not. Dr. Hughes Bennett[1] very aptly remarks—"If by the term inflammation be understood pain, heat, redness, and swelling, or the presence of lymph or pus, then tubercle is not inflammatory. If, on the other hand, we consider that the essential phenomenon of inflammation is an increased exudation of the blood-plasma, then tubercle must be regarded as an inflammatory product." The importance attached to this question, which has engaged the attention of so many eminent men, may be supposed to arise from its bearing upon treatment. It will be shown, however, that in this point of view it is of very little importance, the grand question being whether tubercles arise from a sthenic or asthenic state of the system; inasmuch as inflammations occurring in each of these states demand an almost opposite line of treatment the one from the other.

Nor is it of any practical importance, as Louis has remarked, to ascertain the exact seat of tubercular matter. It has been shown by Guillot, Carswell, and Reynaud to be present in the air-tubes and cells, but it has not been clearly demonstrated elsewhere.

It may be inferred, then—

1. That the deposition of tubercular matter in the lungs depends on a peculiar asthenic state of the constitution, *the tubercular diathesis.*
2. That it is preceded by *local hyperemia.*

CAUSES.

The causes of phthisis pulmonalis may be considered as twofold—one class inducing the tubercular diathesis, and the other favoring the development of tubercles in the lungs after this derangement of the constitution has taken place.

Causes of the tubercular diathesis.

Although we know not the exact nature of those derangements of the system which constitute the tubercular diathesis, we may still expect that observation will enable us to detect the existence of certain antecedent phenomena, which, by their very frequent occurrence, may be considered as causes; in the same manner as we have ascertained that contagion is the cause of certain diseases, although we do not know in what manner it acts on the body.

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[1] Edinb. Med. and Surg. Journal, No. 163.

Many such causes have been brought forward by different writers. Louis, however, has shown, either from his own researches or from those of others, that the greater number of such supposed causes have been admitted on insufficient data. Since the time when I learnt the importance of this inquiry from that distinguished physician, I have had more than four thousand cases of phthisis under my care, and have endeavoured to observe and record all the facts bearing both upon the causes and terminations of this disease. On analysing the results thus obtained it has become apparent that additional observations are required, those of any one person, however extensive they may have been, being insufficient for correct induction. I shall still pursue this inquiry, therefore, in conjunction with other gentlemen, some of whom have been educated in the Birmingham General Hospital, and who are practising in populous neighbourhoods; and I shall reserve to a future time a detailed account of the analysis of the facts hitherto collected, in the hope that when the number of observations shall be more extensive, and shall be joined to those of others, they may lead to results commensurate with the importance of the subject. In the mean time it may be well to state in general terms to what extent the conclusions at which I have already arrived agree with those of Louis.

He admits as causes of the tubercular diathesis, *lymphatic temperament, female sex,* and *continued febrile action;* and he does not deny the influence of *hereditary transmission.* He rejects as special causes, *bad food, impure air, depressing passions,* and other debilitating circumstances; but allows them to be causes of a variety of diseases, of which phthisis is one. He excludes *climate* and *temperature, trade* and *occupation, delicacy of constitution,* and *inflammations of the thoracic viscera.*

My own observations fully confirm the influence of *temperament, sex,* and *continued febrile action;* but they further tend to prove that some causes which are only allowed by Louis to act generally, are in fact special causes of phthisis. These are *mental and physical depression.*

The influenza[1] of 1837 was not accompanied by long continued febrile action, but was characterized by extreme depression of the nervous system. Now the number of cases of phthisis which seemed to owe their origin to an attack of this disease was very great; having occurred in persons who, previously to their suffering from the influenza, had enjoyed good health, and had presented no traces of strumous diathesis. So also chlorosis has very frequently terminated in phthisis, more especially where the periodical menstrual discharge of the female has been arrested. As illustrative of the effects of mental depression, it may be stated that many cases seemed to arise out of an attack of syphilis or gonorrhœa. Several of the young men thus circumstanced had previously enjoyed excellent health, and were apparently free from any kind of constitutional taint whatever. In most of them the symptoms of the venereal affection had not been violent; but living in the constant dread of its discovery by their friends, they suffered greatly from anxiety of mind. This is only one of the sources of mental depression which, in my experience, has been so often followed by phthisis, that I cannot refrain from considering it to be a special cause of this disease.

The influence of hereditary transmission was not very strongly marked in the cases that fell under my notice, if by that expression it is intended to denote that one or both parents had died of phthisis pulmonalis. Although several instances occurred of whole families having successively followed one parent who had died of this disease, yet in the majority of cases phthisis showed itself in one member of a family in which both parents were either alive, or had died of some other complaint. It was not possible, however, to ascertain in all cases whether such parents, as died of other diseases, had tubercles in the lungs or not; nor was information in general obtained concerning the cause of death in relatives more distant than parents or brothers and sisters. It is not sufficiently taken into consideration that as about one fifth of all deaths occur from phthisis, one fifth of all consumptive persons must have been descended from one or both phthisical parents, some allowance being made for those who die before marriage. To establish, therefore, the influence of hereditary transmission, more than one fifth of phthisical persons must have descended from consumptive parents. But if the term hereditary transmission is intended to denote that one or both parents had at some period of their lives presented traces and signs of a strumous diathesis, then its influence has, in my opinion, been by no means overrated.

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[1] Treatise on the Influenza of 1837. By the Author.

It is extremely difficult to estimate the influence of trade and occupation in the production of different diseases for many reasons; and amongst others, because in most cases all the different branches of a trade have been classed under one head, whereas many of them affect the health quite differently from others. Thus there are several different processes in the manufacture of pearl-buttons, but only one or two of them in which the workmen are surrounded by an atmosphere charged with pearl-dust. So in needle-making, the pointing alone gives rise to metallic dust. Owing to the facilities offered by this neighbourhood, a great many facts have been collected bearing on the connexion between trade and disease, from which it appears that in dry grinders, pearl-button turners, and persons following such occupations as raise a fine dust in the atmosphere, all pulmonary diseases, and phthisis amongst them, are more than ordinarily prevalent, but that phthisis does not prevail above other pulmonary disorders, such as bronchitis. On the other hand, it does not appear that the strumous diathesis, as seen in its effects on other organs than the lungs, is at all more frequent in persons following these occupations than in the generality of individuals. So that the facts alluded to tend to prove that the development of the tubercular diathesis is not induced by any particular mode of employment, but that, amongst those in whom it already exists, the deposit of tubercular matter in the lungs is sometimes precipitated.

The results of pleuritis and pneumonia recorded in Chapters XV and XVIII, in connexion with many other facts which might be adduced, tend fully to confirm the opinion of Louis, that inflammations of the thoracic viscera do not induce the tubercular diathesis. It is, indeed, difficult to see in what manner inflammation of a particular organ can induce general constitutional derangements otherwise than by impairing its function, and thereby interfering with the formation or purification of the blood, to which the proper performance of such function may be essential. In this manner inflammation of any organ of the body, particularly of one concerned in the process of primary assimilation, might indirectly favour the development of the tubercular diathesis.

Causes of tubercular deposit in the lungs.

It follows from a consideration of the nature and development of tubercular matter, that all causes which tend to determine blood to any particular parts of the body, must facilitate the deposition of tubercle in those parts, if the tubercular diathesis previously exist. The principal causes which thus act on the lungs are *age, cold applied to the surface of the body, suppression of the menses, certain forms of fever,* and *inflammation of the thoracic viscera.*

Nothing is more fully established than the influence of *age* on the development of tubercle in the lungs. This is in consequence of the blood being more freely determined to the lungs at certain periods of life, more especially about the time of puberty, when the process of respiration becomes very vigorous. For the same reasons, when in early childhood the development of the brain takes the lead, and when for that purpose a great supply of blood is furnished to it, the deposition of tubercle in that organ is a common occurrence. My table of ages corresponds very closely with that of Louis.

The influence of *cold applied to the surface of the body* in producing congestion of the lungs, is a fact of daily observation. It is generally followed by catarrh, which speedily passes off; but if the tubercular diathesis should be strong, such congestion must more or less favour the deposition of tubercle in the lungs. This effect of cold, however, is relative, and not absolute. Thus it may take place both in cold and in warm climates, although it is less likely to occur in those countries where the temperature is equable. We seldom witness the direct termination of such congestions in tuberculization of the lungs; because, in the great majority of cases, the congestion is speedily removed by copious expectoration. When it does thus terminate, it is generally after repeated attacks.

Pulmonary congestion resulting from the *suppression of the menstrual discharge* is of frequent occurrence; no state of the system has, in my experience, more frequently preceded tuberculization of the lungs. Such was the case in a large majority of phthisical females between the ages of 16 and 30. In most of these cases there was also chlorosis, so that both the cause of the diathesis and of the local determination were present. Hence the paramount importance of arresting by an appropriate treatment the progress of such cases whilst yet in an early stage.

In *fever* also both causes are in action; the low state of the system inducing the diathesis, and the pulmonary congestion which is usually present, favouring its characteristic deposit in the lungs. A large number of cases of phthisis were most clearly traced to this source.

The direct influence of *inflammation of the thoracic viscera* has been denied by Laennec and Louis. The soundness of their deductions, in this respect, is fully confirmed by my own researches. There appear to be some obvious reasons for this. The most common form of such inflammations is bronchitis. In this disease plastic lymph, which is so constantly associated with tubercular development in the lungs, is scarcely ever thrown out. Although, therefore, the secretion which takes place from the air-tubes during bronchitis, occurring in persons who labour under the tubercular diathesis, is, doubtless, modified thereby in some degree, yet the liquid form of secretion is more or less preserved, and being expectorated, does not form tubercular masses. This disease is rather the scrofulous bronchitis of Dr. Graves. Even if plastic lymph were deposited in the bronchial tubes, it would easily be expelled before it could be transformed into tubercle or become a nidus for it, unless, as in Reynaud's case above referred to, it filled the whole tube. The secretion of the air-vesicles, however, is often of a more plastic nature, as is seen in certain cases of pneumonia; and hence tubercle is more likely to supervene on this latter disease than on bronchitis. Even here, however, in a very acute attack, the exudation being copious and moist, would either be speedily converted into pus and destroy the patient, or would be quickly reabsorbed. Hence it is that acute pneumonia is seldom followed by tuberculization of the lungs. The subacute and chronic forms in which plastic exudation predominates are those which might be expected to terminate thus. And this accords with my experience; for it has been seen in tracing out tubercular development, that chronic pneumonia not unfrequently terminates in phthisis pulmonalis, whether it has originally arisen from unknown causes, or from the irritation excited by the presence of an aneurism of the aorta, or other abnormal growth.

CHAPTER XX.

TERMINATION OF PHTHISIS PULMONALIS.

The possibility of phthisis pulmonalis terminating otherwise than by death has been questioned by some; whilst others, arguing from certain appearances found in the lungs of persons who have died from a different complaint, have asserted that cases of recovery from this disease are of frequent occurrence. It will be desirable therefore to ascertain, from an examination of cases accurately observed, whether recovery has ever taken place after tubercular matter has been deposited in the lungs; and if so, at how late a stage of the disease, in what proportion to the number of deaths, and under what circumstances, such recoveries have taken place.

If solid whitish nodules, of the consistence of condensed cellular tissue or fibro-cartilage in the lungs, are to be considered as denoting the previous existence of tubercular deposit, in that case the disappearance of tubercle must be allowed to be a very common occurrence. Rogée,[1] Boudet,[2] and Dr. Hughes Bennett[3] found them either with or without calcareous concretions in from one to two thirds of the bodies they examined. These appearances, however, are found in the lungs of persons who never exhibited the slightest symptom of phthisis during lifetime; and the probability has been shown of their having been sometimes the result of plastic pneumonia. It has been justly remarked by Andral, that unless a bronchial ramification can be traced into these substances, the pre-existence of a cavity cannot be deduced. This, however, rarely happens, having only occurred in Case CVI in my own experience, except in lungs in which unhealed tubercular cavities actually existed at the time of death, and then only in two instances. I have myself only met with a complete open cavity in lungs which presented no trace of yellow tubercle, in Cases CVII and CVIII.

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[1] Archives Générales de Médecine, vol. v.

[2] Comptes rendus, vol. vi.

[3] Edinb. Med. and Surg. Review, No. 163.

Calcareous masses of variable size have not unfrequently been found in the lungs, more especially by Rogée in his cadaveric inspections at the Salpêtrière. Louis very justly remarks, that, supposing such substances to be the residue of tubercles, the symptoms dependent on them could not have been of a very serious kind; and the same remark would apply to the fibrous nodules above alluded to. As it is by no means rare to find a few tubercles scattered through different organs of the body which had not given rise to symptoms during life, so it is not unreasonable to suppose that in some cases tubercles may, in an improved state of the constitution, have been converted into these cretaceous and calcareous masses. But it can hardly be said to have been demonstrated that these deposits have always been preceded by tubercular matter; for in like manner as the calcareous deposit takes place around fibrinous concretions within the heart, and around coagulated lymph in other parts of the body, so the same may take place in the lungs, without such lymph being connected with tubercles.

Be this as it may, it can hardly be disputed that cadaveric inspection alone has proved that tubercular matter may be deposited in the lungs, and be replaced or surrounded by matter of cellulo-fibrous, fibro-cartilaginous, or calcareous consistence, without having inflicted any notable injury on the health. This fact is of the highest interest in regard to the possibility of recovery from phthisis; but the question remains to be solved whether such recovery ever takes place after decided signs of tubercular deposit have been manifested in lifetime.

Many cases might be adduced in which, after unequivocal signs of solid deposit at the summit of one lung, either the physical signs of disease have disappeared, and complete recovery has taken place, or else in which some chronic catarrh alone has remained, and some coarseness of the pulmonary sound over the affected part. It may be said that there is no proof that tubercles had ever been deposited in these cases. In most of them it is probable that the solidity of the lung was occasioned by the presence of coagulated lymph, as in one of the lungs in Case CIII, and that there had been no tubercular deposit. But when it is remembered how many cases of fatal phthisis have commenced with the same train of symptoms, it is hardly too much to assume that several of these, had they not been arrested, would have terminated in a similar manner; so that in such cases the disease may be said to have been prevented rather than cured. Few will hesitate to admit the existence of tubercular deposit in the following case.

Case CV.

Phthisis arrested.

A married lady, æt. 26, the mother of two children, had usually enjoyed good health until about six months ago; when, after some mental excitement, she was seized with a shivering fit; she was feverish for a day or two, when a cough set in, accompanied from the first with expectoration of frothy, clear mucus, which soon became greenish and opaque. For the last month she perspired copiously for two or three successive nights. When seen, she complained much of cough and of some pain about the right scapula. She was frequently troubled with nausea, and occasionally vomited a little sour water or bile. She had slight gastrodynia, and was much troubled with leucorrhœa; the menstrual secretion was rather scanty and pale. She had no hemoptysis. A shade of dullness on strong percussion was perceived under the right clavicle. In this spot there were heard some large moist crackling on deep inspiration, and slight vocal resonance. For several weeks the physical signs remained much the same, when she took a fresh cold, as she thought; and the cough, which had been relieved, reappeared in an aggravated form, with increased expectoration. The crackling was replaced by a circumscribed mucous rattle, and clear pectoriloquy and ringing cough were heard in the same spot with it. She became much emaciated, and had frequent night perspirations. She was then sent to the sea-side, and ordered at the same time counter-irritation on the chest, tincture of sesquichloride of iron and morphia, with a generous diet, including porter. In fourmonths she returned greatly improved. The leucorrhœa had nearly ceased, and the menstrual secretion was natural. She had an excellent appetite, was in good spirits, and had regained much flesh. The cough still existed, but was much less troublesome; the expectoration consisted of clear gray mucus. There was no dullness under the clavicle, and no moist rattle or crackling. In this spot the pulmonary sound was rather hollow and coarse, although feeble, and prolonged during expiration. This improvement continued for some months, when it became evident that she was pregnant. She had a favorable accouchement, and has remained much in the same state for the last seven years, only suffering from cough on taking cold, to which she is rather liable. She may now be said to be a delicate person, but to enjoy an average share of health.

Many persons would be disposed to attribute the suspension of the disease to pregnancy. Such an inference would not be justified by other cases of this kind which I have seen. In none other has the progress of phthisis been arrested by gestation; whilst in many it has run a frightfully rapid course immediately after delivery; and has, on the whole, appeared to reach a fatal termination sooner than it probably would have done had not impregnation taken place.

We still want the confirmation which would be furnished by the inspection of the body of a person who had, during lifetime, presented signs of a tubercular cavity and of its obliteration, and had eventually died of some other disease. This is seen in the following case.

Case CVI.

Phthisis arrested—Death from perforation of the duodenum—Cicatrix in the lung.

A clerk, æt. 24, in the employment of a gun manufacturer, had been in delicate health twelve months, during which period he had been troubled with cough and slight expectoration of yellow phlegm, occasionally streaked with blood. Two months ago (February, 1837) he had the influenza, since which time the cough and expectoration had much increased. A fortnight since he had, for four successive days, rigors in the evening, succeeded by burning fever and copious perspiration in the night. When admitted a patient of the Birmingham

Dispensary, he complained of great constriction of the chest. His bowels were regular, his pulse was of moderate force, 90; his tongue was slightly coated, his appetite was variable, and the expectoration chiefly consisted of thick clear mucus.

Percussion elicited no dullness of sound. The pulmonary sound was loud and coarse under each clavicle. A feeble mucous rattle was heard over the middle of the right side of the chest, both before and behind. Under the left clavicle slight bronchophony was detected. For a month no marked exacerbation of symptoms occurred; but the pulse gradually increased in frequency, and he had several slight attacks of hemoptysis. At the end of that time the pulse was 130, small and sharp. The expectoration was muco-purulent, and contained small, cheese-like masses. He was greatly emaciated, and had profuse night perspirations. Slight, but well-marked dullness was found over a very limited space under the middle of the left clavicle. In the same spot a circumscribed mucous gurgling was heard, a ringing splash on coughing, and clear bronchophony. He was now ordered to leave his employment and to retire to a neighbouring village. He was placed on a generous but unstimulating diet, and summer having commenced, he was enjoined to pass the greater part of the day out of doors. Counter-irritation was employed under the left clavicle, and a mild tonic mixture with hydrocyanic acid was administered. In a short time an amendment took place, the night perspirations abated, the expectoration became gradually clearer and gray, without any traces of blood in it, and finally ceased; the dullness nearly disappeared, and a mucous click was all that remained of the gurgling. He became driver of a milk-cart.

In six months more every trace of disease vanished. He regained his flesh and lost his cough. His pulse was moderately full, 72. His appetite was good. On examining the chest no dullness, rattle, or resonance of the voice could be detected; but the pulmonary murmur seemed to be a little coarse under the left clavicle.

After a lapse of two years he sought my advice, complaining of indigestion, with a sense of fulness and sometimes of pain at the stomach, frequent sour eructations and flatulence. His bowels were rather confined, and his tongue was covered with a thick yellow fur. The pulse was moderate. He had resumed his previous occupation as a clerk. Mild alterative medicines were ordered him. The next morning, feeling himself better, he went to his employment, and his bowels were twice opened, when, at one o'clock, he was suddenly seized with excruciating pain in the bowels, which persisted, unrelieved by the remedies administered by Mr. Meek, who was called to him, up to six o'clock the next morning, when I was consulted. His pulse was hardly perceptible, the extremities were cold, his expression of countenance was anxious, and his face pallid and clammy. The abdomen was much swelled, the highest part as he lay sounding clear and tympanitic, and the more dependent parts dull; when changed from one side to another, the lower side became dull and the upper clear. It was tender to the touch, but not remarkably so He was evidently sinking, and died at ten o'clock, a.m., twenty-one hours after the sudden invasion of the abdominal pain.

Inspection, in the presence of Mr. Meek and Mr. Francis Galton. On opening the abdominal cavity, the mesentery and intestines were seen to be highly injected, and several patches of yellow lymph were lying on different portions of the peritoneum. A considerable quantity of brown-coloured fluid, with a fecal odour, was found in the abdomen. An opening was seen in the serous coat of the duodenum, within half an inch of the pylorus, which measured about three lines by two. The mucous membrane of the stomach was pale and softened, and was highly mammillated along the great curvature. At the part of the duodenum corresponding to the external opening was a deep ulcer, with hard, white, thickened edges, about the size of a fourpenny-piece. Nearly opposite to this was a similar ulcer of a smaller size, which was prevented from communicating with the abdominal cavity by the adhesion of a small gland. The liver was pale, hard, and of close texture.

The heart and the right lung were both healthy. In the front of the left lung, about two or three inches from its summit, there was an appearance of deep puckering, which felt hard and lumpy, and around which the lung was of a much blacker colour than elsewhere. On cutting into it a fibro-cartilaginous plate was found about one inch in length and half an inch in width across its centre; and, at one part, two lines in thickness. Three white lines radiated from it, one of which was traced into a bronchial tube. A little distance off, a small whitish nodule was discovered of fibrous consistence; and lower down, three chalky masses of a cream colour, and of the size of rape seeds, were found surrounded by healthy lung.

It is hardly possible to conceive a more complete proof of restoration to health, after the formation and subsequent healing of a tubercular cavity in the lungs than is here afforded. In the cases hitherto considered, the extent of lung diseased has been small, and this may be remarked in most of the cases in which phthisis has been arrested. Three or four have presented traces of extensive disease, but the amendment observed in them has only been temporary. They have either died, or else still labour under phthisis pulmonalis in a very chronic form.

The following cases furnish remarkable exceptions to this rule.

Case CVII.

Phthisis arrested—Death from hemoptysis.

A gentleman's coachman, æt. 22, whilst in Scotland four months ago, caught cold and felt "stuffed" at his chest. A month after this a cough set in, accompanied by clear white expectoration. He was bled, and placed on very low diet. On coming to Coventry he was seen by Mr. Troughton, and on returning to his friends in Birmingham he fell under my care. He was very hoarse, and had been so for three months. He had severe night perspirations during three weeks. He was greatly emaciated, and could not walk without support. He had latterly expectorated sputa of a yellowish and dark gray colour, sometimes streaked with blood. His pulse was 130, very small and feeble. From the left clavicle down to the nipple the chest sounded as dull as possible. Immediately below the left clavicle, mucous crackling and imperfect pectoriloquy were heard. He was ordered cod-liver oil and a generous diet. For three weeks he improved rapidly, and gained much strength and flesh; but he had frequent night perspirations, and expectorated muco-purulent fluid, which seemed to contain cheesy tubercle. The cod-liver oil purging him, it was discontinued, and quinine, with a morphia linctus, was given to him. Four months after this he had become quite plump and strong, and could walk ten or twelve miles a day. His cough was occasionally troublesome, but he only expectorated greenish mucus. There were still dullness on percussion, and absence of respiratory sound over the upper half of the left lung, but no rattle. One day, whilst walking, he was seized with hemoptysis, and suffocated.

Inspection. The colour of both lungs was almost white, spotted with numerous patches of a bright red colour, with fringed edges like a pink. The right lung was enormous. The left lung was much shrunk, and was everywhere closely adherent to the chest. Its upper two thirds were converted into a mass of gray induration, which presented a granulated surface on incision. Exactly at the apex was a large cavity lined by a smooth membrane. A small cavity existed at the apex of the right lung, surrounded by clusters of gray granulations fused together. In an angle of this cavity was a small quantity of yellow tubercular matter, which was all that was found in any part of the body.

It would appear that this patient was seized with acute pleuro-pneumonia, which subsequently passed into a chronic state. In the meantime, the tubercular diathesis was developed, and a portion of the gray matter in the indurated lung became the seat of, or was converted into yellow tubercle, as in Cases VII, XXIII, CIII, CIV, &c. This having softened, cavities were formed, with the usual concomitant symptoms, and death seemed to be rapidly approaching, when another change of the constitution occurred; the assimilating organs were restored to healthy action, and wholesome blood was formed; a change which was manifested by the returning plumpness, strength, and general health of the patient. From that moment no more tubercular matter was formed in the lungs, and that which was already there, having been got rid of by expectoration, the progress of the disease was arrested.

If the effusion of gray matter had taken place around the tubercular cavity subsequent to its formation, it would have closed in upon it, and have squeezed its sides together, as is seen in similar cases. The cavity being in an open state, is of itself an evidence that it was formed out of the gray mass, which was too extensive and too solid to admit of contraction around the cavity.

The source of the fatal hemoptysis could not be traced, but it was doubtless caused by the rupture of a blood-vessel, which had been affected by ulceration of the lungs. It is impossible to conjecture how long the patient might have lived had it not been for this accidental occurrence; for he had regained health and strength; and the enormous size of the right lung must have enabled it, in conjunction with the lower lobe of the left lung, to carry on the respiratory process in a degree fully proportioned to the wants of the system.

The extent of lung rendered impervious to air by chronic pneumonia was very considerable, and the portion of it which had been actually destroyed by tubercular ulceration was much greater than that in Cases XXIII and CIII, or than the physical signs denoted in any of the arrested cases of phthisis hitherto observed by me.

Case CVIII.

Gray granulations and hepatization—Cavities—Bronchitis.

A single lady, æt. 23, had suffered for twelve years with some dyspnœa and cough. Four years ago she was under the care of Mr. Alfred Baker and myself, with symptoms of phthisis, at which time she expectorated yellow matter, occasionally streaked with blood. On two different occasions there were faint signs of tubercular deposit at the apex of each lung. She improved, however, remaining apparently asthmatic, and expectorating clear mucus. Two years since she had a severe attack of hemoptysis, and again one month since a slight attack. When visited she was very thin, and her appetite was bad. She had a troublesome cough, with expectoration of clear mucus mixed with pearly gray lumps. She had much dyspnœa. There was a mixture of bass-viol and cooing sounds all over the chest, with some small muco-crepitant rattle; but the chest sounded clear on percussion. A week or two after this she took cold, and was seized with a very violent attack of bronchitis. Fine muco-crepitant rattle was heard all over the chest, and a trace of dullness was perceptible under the left clavicle. The sputa were scanty and very tenacious. After a slight degree of improvement she relapsed and rapidly sank.

Inspection. The whole of the left lung and the apex of the right lung adhered to the chest. At the summit of the left lung was a cavity which would have held a walnut, with anfractuous walls of a dark slate colour. Around this and for some distance down the lung were patches of gray induration, and agglomerated granulations. The back part of the lower half of this lung was affected with static pneumonia. There was a similar but much smaller cavity at the apex of the right lung, the remaining part of which was rather œdematous. The lining membrane of the bronchial tubes was red, soft, and swollen. They contained much adhesive mucus. There was not a trace of yellow tubercle in any part of the body. The heart was healthy.

This case resembles the last in the circumstance of the formation of yellow tubercle having entirely ceased after cavities had been formed from which it had been evacuated.

From these cases it is clearly deducible that health may be restored, and every trace of tubercular matter may disappear after it has been deposited and given rise to cavities in the lungs.

The different processes by which cavities are healed and tubercular matter is rendered inert in the lungs are fully discussed in Hasse's 'Pathological Anatomy;'[1] but, as has before been remarked, it is probable that many of the puckerings of the lungs stated to have been the result of tubercular deposition have, in fact, been produced by that of gray matter which has never been tubercular.

Although great exertions have been used to ascertain the proportion of cases in which the disease was arrested to those in which it proved fatal, it has been found impossible to arrive at any certain results. All that can be stated at present is, that out of nearly 4000 persons supposed to labour under phthisis, treated between the close of 1835 and the commencement of 1846, thirty-one survived at the latter time, in whom the most unequivocal signs of a tubercular cavity in the lungs had been observed, either by Mr. Alfred Baker, late house-surgeon of the Birmingham General Hospital, or by myself. Of ten of these no more is known than that they have been reported by their friends as living. The other twenty-one have been seen by me within the last two years.

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[1] Dr. Swaine's Translation, Syd. Soc. Ed. p. 338.

Of these, eight still labour under the disease in a chronic form, four are tolerably well, but expectorate muco-purulent fluid, and have signs of a cavity remaining. The other nine enjoy an average amount of health, have slight cough, with expectoration of pearly gray mucus, and have lost all traces of a cavity. There is in most of them, however, coarse and feeble pulmonary sound in those situations where a cavity had existed.

There is a great difficulty in determining the proportion of recoveries to deaths, which is rendered almost insurmountable by the impossibility of following many of the hospital patients up to the termination of their illness. Nor can we ascertain how often the disease is arrested in its early stages, before tubercular softening has taken place ; but I feel well assured that this occurs much more frequently than is generally supposed, and hence the appearances so often found in the lungs, and which, in many cases at least, must have been caused by the presence of tubercles. Taking the most favorable view, however, the proportion of recoveries to deaths is so small that the difference in the circumstances under which these opposite results take place can only be satisfactorily ascertained by the collation of a large number of cases collected by many different persons.

The following facts, however, are not altogether devoid of interest, and may be useful in connexion with those collected by others.

Of the persons who survived in 1846, three fourths were between the ages of 35 and 45, and only two were under 20, a fact tending to confirm the opinion that the chances of recovery increase with age. It has been long known that the disease runs a more chronic course in proportion as the patients advance in years, and it is therefore probable that recovery takes place more frequently at this period of life, in consequence of time being allowed for the removal of the constitutional derangements upon which this disease so peculiarly depends.

It cannot be said that any one of these persons actually wanted the necessaries of life, for nearly the whole of them were so far in easy circumstances that they could procure everything that was ordered for them, and were enabled to obtain a change of air.

Of those who were watched from first to last, the amendment commenced in one half when they were at a greater or less distance from home in pure air, generally at the seaside.

Whilst under my care they were all treated, as were, indeed, the whole four thousand, on the tonic and sedative plan presently to be detailed. It is but fair to add, that in one case amendment followed the employment of active remedies prescribed for an attack of acute pleurisy.

CASE CIX.

Phthisis arrested—Acute pleurisy.

An upholstress, æt. 23, in consequence of severe trials, had fallen into a low state of health, and was greatly depressed in spirits. She was troubled with a dry cough, was restless and feverish, and lost her appetite and flesh. At this time she got wet through, after which her cough became aggravated, and she had two or three attacks of hemoptysis, in one of which about four ounces of blood were brought up. When examined she complained of flying pains in the chest and of troublesome cough. She expectorated muco-purulent fluid which contained some cheesy masses, and was sometimes streaked with blood. She had profuse night perspirations, was emaciated, had no appetite, and was very feeble. The left side of the chest sounded well. There was considerable dullness for three inches below the right clavicle. In the same spot were heard circumscribed mucous rattle, a small splash on coughing, and clear pectoriloquy. The rest of the lung seemed healthy. In less than a week after this she was seized with a very acute attack of pleurisy on the right side, accompanied by intense pain and catching respiration, great fever, and a sharp pulse. Twenty leeches were thrice applied, followed each time by large linseed poultices, and afterwards mercurial ointment was rubbed in. On her recovery from this attack the dullness under the right clavicle was found much diminished in extent; hollow respiratory sound with an occasional click had replaced the mucous rattle; pectoriloquy remained. The quantity of expectoration had greatly diminished. Three months from this time she had regained much of her original plumpness, felt strong, and had a good appetite. She expectorated only a grayish mucus occasionally tinged with yellow, but never mixed with blood. The dullness under the clavicle had still further diminished in extent. Vocal resonance was but slightly marked, and was rather buzzing. The respiratory sound was feeble and coarse over the same spot, and was prolonged during expiration. She has now been married nine years, and remains much in the same state, occasionally taking cold, when there is a slight aggravation of symptoms.

It can hardly be said that antiphlogistic measures were fully adopted in this case, for venesection was not practised at all, and the moment the acute inflammatory symptoms were subdued the tonic regimen was resumed.

The disease is generally hurried to a fatal termination on the occurrence of perforation of the pleura. This accident can, in most instances, be easily recognised by the signs described by Louis. He found the exact moment of perforation marked in all his cases by sudden pain, like that which usually occurs in perforation of the intestines. I have, however, met with exceptions to this rule, both in the chest and abdomen.

Case CX.

Phthisis—Perforation of the pleura.

A patient of the Birmingham Dispensary, a carpenter, æt. 28, descended from consumptive parents, enjoyed good health until he received a violent kick on the right side of his chest, which gave rise to an attack of pleuritis, succeeded by cough and dyspnœa, which have persisted. When visited, in company with Mr. C. Gem, he stated that he had not been able to lie on his left side for many months, and that latterly he could not lie down at all; that, for some time, he had expectorated yellow phlegm, but that now it had changed into thick mucus, which contained black colouring matter. A fortnight since he felt something suddenly snap in his right side, and a day or two afterwards, when he moved, he heard a noise in his chest like the "ringing of a bell," which was heard also by the bystanders, who likened it to the shaking of water in a bottle. He was bolstered up almost perpendicularly, inclining to the right side. His respiration was hurried, his countenance anxious, and his pulse small.

Immediately below the left clavicle there was a shade of dullness on percussion, as compared with the middle of the same side. The lowest part of this side sounded dull both before and behind, varying with position, being dull in the most dependent parts. Under this clavicle the pulmonary sound was rather coarse and prolonged during expiration. Over the middle of the chest it was intense, and at the lower part it could not be heard. On the right side, both before and behind, an extremely clear and ringing sound was heard on percussion, from the summit to a distance of two inches below the clavicle, when the sound became suddenly very dull. Immediately under the clavicle and also up the spine, some coarse but very feeble pulmonary murmur was heard; in other parts of the right side no sound could be detected; but when he coughed, spoke loud, or moved himself, a noise was audible resembling two or three tinklings of a bell. On a level with the nipple there was no difference in the measurement of the two sides of the chest; but a little below this spot, the right side was three inches larger than the left. In two days he died, and a whey-like fluid escaped from his mouth during his last moments.

Inspection. The left pleural cavity contained half a pint of bloody serum. There were many gray granulations, and small yellow tubercles in the upper portion of the left lung, around which the pulmonary tissue crepitated on pressure. The lower lobe was very voluminous, and quite healthy. The whole of the right pleural cavity was lined with a coating of soft lymph two lines in thickness. It contained five quarts of lemon-coloured serum, which, as it was laded out, became turbid from the mixture of pus and flocculi of lymph, which had subsided. The right lung was compressed against the spine, and measured eight inches in breadth, and from one to three inches in depth. From a point in the costal pleura, a little below the humeral end of the clavicle, a round band or false membrane extended inwards, backwards, and downwards, to the collapsed lung, thus traversing the pleural cavity. Within an inch of the spine, on a level with the third rib, was a small opening in the pulmonary pleura, with rounded edges, which communicated with a cavity lined by a false membrane, and this was connected with a larger cavern, into which a good-sized bronchial tube opened. Air blown into the trachea, before the lung was cut into, passed into the pleural cavity, through this opening in the pleura. The left lung, on being inflated, was found to contain several tubercular cavities, the walls of which were ragged, and also many small tubercles and gray granulations.

The moment of perforation was graphically described by the patient; but he distinctly stated that the snapping was not accompanied by pain, or followed by any great dyspnœa. This may be easily accounted for. Pleurisy, with effusion, had existed some time on this side, so that the pleura thus coated with lymph, had become insensible to pain, which is usually produced by the contact of the contents of a tubercular cavity with healthy pleura. So also the lung, having been previously compressed by pleuritic effusion, could not be suddenly condensed to any great additional extent by the communication established between the external air and the pleural cavity, more especially as it was prevented from entirely collapsing by the band that connected it with the side.

Thus, too, in Case CIII, although the perforation of the intestine was marked by sudden prostration, no characteristic pain was experienced, for there the peritoneum was covered with lymph in the same manner as was the pleura in this case. However, as the existence of chronic peritonitis was known, the perforation was recognised in the absence of severe pain, its most marked sign under ordinary causes. These cases are merely brought forward as exceptions which ought to be known.

In every other case of perforation of the pleura or of the small intestine which has come under my observation, sudden acute pain has been present.

Louis has been unable to trace any connexion between the amount of disease in the lungs and the duration of life after perforation of the pleura has taken place. The following considerations show that such a connexion can only be discovered by separately estimating the disease of each lung. For when death speedily follows perforation of the pleura, it results from the sudden collapse of the perforated lung. Now, the more extensive the tuberculization of this lung is, the less is there left of its parenchyma which can collapse, and therefore the slighter is the shock inflicted on the system, and the chance of speedy death is by so much lessened. Again, a similar effect is produced when the lung is prevented from collapsing by adhesion to the walls of the thorax. The immediate danger having been thus surmounted, all other things being equal, the duration of life must depend on the soundness of the opposite lung, in which is then concentrated the whole process of respiration.

Viewed in this light, an analysis of Louis' cases reveals an intimate connexion between the disease of the lungs and the duration of life. Thus, in four of his cases, in which death took place in less than three days from the time of perforation, the opposite lung was diseased throughout. Whereas in two other cases, in which life was respectively protracted to eighteen and to twenty-three days, merely the summits of the unperforated lungs were found tuberculated when the bodies were inspected, and in all probability were still less diseased at the time when perforation occurred.

The examination of seven cases in my own practice has been attended with similar results. Of these the following case is very interesting.

Case CXI.

Phthisis pulmonalis—Speedy death from perforation of the pleura.

A labourer, æt. 18, had for some time suffered from cough, and a severe pain across the middle of the chest, attended with profuse night perspirations and a sensation of heat in the palms of his hands and the soles of his feet. A month since he had an attack of diarrhœa, which left him much emaciated. At the time of his admission into the Birmingham General Hospital he complained of cough, which deprived him of rest, and which caused him pain in the chest when it was severe. His face was pallid, and he had an anxious expression of countenance. He perspired profusely at night, and expectorated a large quantity of gray mucus, which was occasionally streaked with blood. His appetite was good, his tongue pale and flabby, and the pulse small and frequent; his respiration was hurried, 38. The chest was nowhere very resonant, but the sound was duller on percussion under each clavicle than elsewhere. The pulmonary sound was generally coarse, more particularly at the summit of the lungs, where it was prolonged during expiration, and under the right clavicle, where it was also hollow. There was slight vocal resonance under both clavicles.

He was much relieved by a morphia linctus, quinine mixture, and generous diet.

On the morning of his death, which occurred four weeks after his admission into the hospital, he seemed as well as usual, and was talking to another patient, when he was suddenly seized with intense pain in the right side of the chest, and great dyspnœa, and died within a quarter of an hour, gasping for breath.

Inspection. On opening the thorax a large quantity of air rushed out of the right pleural cavity. There was no fluid on either side, and no adhesion except a slight old band, which connected the back part of the apex of the left lung with the walls of the chest. The right lung had shrunk to three fourths of its natural size. On inflating it through the trachea, air was found to escape through a small aperture in the upper and outer part of it, corresponding to the axilla, which communicated with a cavity capable of containing a walnut, filled with thick grayish-yellow matter, and into which a large bronchial tube opened. Throughout the rest of the lung, which to a certain extent crepitated, and was considerably augmented in size by inflation, a great number of gray granulations and yellow tubercles were thickly scattered. The left lung was most densely studded with these bodies, and at the back of its apex, opposite to the adhesion mentioned, there was a small cavity of the size of an almond, full of the same thick matter.

The amount of healthy lung on the side opposite to the perforation was so exceedingly small that it is no wonder that the patient was soon suffocated, as it were, after the collapse of the other lung.

In estimating the effects of perforation on the duration of life, Case CX can hardly be taken into consideration, for the right lung had been so completely compressed by the pleuritic effusion that the entrance of the air on perforation taking place could produce little or no effect on the respiratory process, which was already confined to the opposite lung.

The prognosis, therefore, on perforation of the pleura taking place, must depend on the state of the *opposite* lung, and must be favorable or otherwise in a direct ratio with the soundness of that lung.

It occasionally happens that complete recovery takes place after this accident, in which case the perforation probably has been produced by a very small amount of tubercular deposit in the lung, or by some other cause not connected with phthisis, as is seen in the following cases.

CASE CXII.

Perforation of the pleura with displacement of the heart—Recovery.

A man, æt. 39, of middle stature, slender conformation, and strumous diathesis, by trade a rule-maker, enjoyed a good state of health previous to the 12th of June, 1843, when he was suddenly seized with an acute pain on the left side of his chest, which obliged him to leave work, and to seek my advice.

When I arrived he was in bed. The symptoms were pain in the left side; cough and dyspnœa without febrile symptoms. Percussion elicited a preternaturally clear sound over the whole of the left side of the chest, over no part of which could any pulmonary sound whatever be detected. On forced respiration a metallic tinkling was audible, and, when speaking, a resonance of the same character. The pulmonary murmur was puerile over the right side, the impulse of the heart was very distinct on the right of the sternum, as were its sounds; its presence in its new situation was also corroborated by percussion. The measurement of the chest showed a difference of an inch and a half in favour of the left over the right side. He had some mucous expectoration, and continued ill during several weeks, and was much emaciated. Under the exhibition of iron and alteratives he was enabled to return to his work in nine weeks, when the respiratory murmur became distinct over the whole of the left side, the heart resumed its natural position, and the left side became somewhat flattened, and contracted so as to measure an inch and a quarter less than the right side.

Signed, C. Meek.

I saw this patient during his illness, and can verify the accuracy of the description of the physical signs. He remains quite well.

Case CXIII.

Pneumo-thorax, without previous symptoms of phthisis or pleurisy.

A healthy-looking servant girl, æt. 16, without the slightest trace of strumous diathesis, was admitted into the Birmingham General Hospital, labouring under primary ulceration of the cartilages of the right hip, produced by a fall. She was in excellent health, and had menstruated once or twice, but not subsequent to her admission into the hospital, six or seven months from the time of the accident. After six months' residence there, the state of the hip had very much improved, and her health continued very good, when she complained of a troublesome cough, accompanied by expectoration of clear mucus; and, on being questioned, she stated that she had coughed occasionally for four months. Three days after this, whilst coughing in the night, she was suddenly seized with severe pain at the bottom of the right side of the chest. The next day she was feverish, and complained of thirst. Her skin was hot and dry, the pulse 112, quick and sharp. The tongue was white. Respiration was hurried, and the cough distressing. She lay on her left side. The whole of the right side, more particularly the lower half, sounded much clearer than the left side. The respiratory sound consisted of a hollow blowing over the right side, except up the spine and under the clavicle, where it was blowing and coarse, but not hollow. Clear metallic tinkling was heard during the act of speaking over the whole of this side, especially around the nipple and near the lower angle of the scapula. Over the left side the pulmonary sound was pure and intense. The action of the heart was jerking, and its sounds rather sharp. The right side, on a level with the nipple, measured an inch more than the left side. Two days after this her friends removed her to her own home, but in fourteen days called to report her recovery from the chest affection.

CHAPTER XXI.

DIAGNOSIS OF ACUTE PHTHISIS PULMONALIS.

Cases of acute phthisis in which life is destroyed within eight or nine weeks by the development of tubercles and gray granulations in the lungs, without the formation of cavities, are rare, and have hitherto been considered to be very difficult of discovery. I have only met with twenty cases of this description.

In most of these both lungs were nearly filled with gray granulations and yellow tubercles, varying in size, from that of a small pin's head to a rape seed. Many of the granulations had yellow specks in them, and some of the larger yellow tubercles were soft, and in one instance (Case cxvi) a few of them had been expectorated.

In the following case there were no single gray granulations, but merely some masses of gray induration.

Case CXIV.

Acute phthisis.

A plater, æt. 36, enjoyed good health until he was attacked with a dry cough, which, in five weeks, was aggravated, and was accompanied by dyspnœa, and occasional shivering fits. After two weeks more he became a patient in the Birmingham General Hospital. He was emaciated, his face was rather livid, and covered with a profuse perspiration. He complained of slight pain under the right nipple, and generally lay on his back. The dyspnœa was extreme, and there was great anxiety of countenance. His respiration was 40 in the minute. The pulse was full, but very compressible, 130. His cough was frequent, and was accompanied by the expectoration of muco-purulent fluid.

There was some dullness on percussion immediately under the right nipple, and towards the outer edge of the right side of the chest. About this spot a fine moist crepitation was heard, and over the whole of the chest there was a mixture of mucous rattle, bass-viol and cooing sounds. In three days' time he became delirious, the dyspnœa being most distressing, and his face very livid. He died in two days more, eight weeks from the time when he was in perfect health.

Inspection. The apex of the left lung adhered firmly to the thorax. The substance of the lung was everywhere redder, and somewhat heavier and more friable than natural, but still crepitated on pressure. It was studded with a great number of opaque yellow tubercles, from the size of a mustard seed to that of a rape seed, some of which were surrounded by semi-transparent gray induration. The lining membrane of the trachea and larger air-tubes was redder than natural, and there was a small ulceration in the larynx. Two or three glands of the mesocolon were enlarged, and contained cheesy tuberculous matter. At the opposite points in the colon there were ulcerations of the mucous membrane, which was, for some distance, red and soft.

This patient, having enjoyed excellent health to within eight weeks of his death, when cough set in accompanied by shivering fits, it is improbable that the lungs were previously diseased; so that all the isolated granulations were both deposited and transformed into yellow tubercle in this short time, or else tubercles were formed without granulations in the midst of the healthy lung. The latter is the most probable supposition.

Some traces of inflammation either of the substance of the lungs, of the lining membrane of the bronchial tubes, or of the pleura occurred in every case. In none, however, were there signs of a high degree of inflammation. Thus the evidences of pleurisy consisted for the most part of adhesions between the pleural surfaces; in one case only some thin films of lymph were found lying on a portion of one lung. Pneumonia was in general limited to the first stage, and resembled the congested state of the lung observed in typhus. When the second stage was reached, only a patch or two was seen, as in Case CXVII. Louis seems to have found a large amount of pneumonia complicating his cases. The injection of the bronchial membrane was either limited to the large tubes, as in Cases cxiv and cxvii, or was of that deep violet colour found in typhus, or in some forms of heart disease, where the symptoms during lifetime have indicated either congestion or a chronic form of disease, rather than acute sthenic bronchitis. In short, a low degree of inflammation seemed to be present, of somewhat the same intensity as that which may be supposed to have been concerned in the process of throwing out the excess of blood-plasma, from which tubercle and lymph are formed in the lungs of phthisical persons.

Age. Of the twenty cases of acute phthisis which have come under my observation, seven cases occurred between the ages of 6 and 10, two between 10 and 20, eight between 20 and 30, two between 30 and 40, and one above 70. This last age is remarkable.

Case CXV.

Acute phthisis—Pericarditis.

A labourer, æt. 73, accustomed to much exposure, and who had enjoyed good health, with the exception of a winter cough and occasional flying rheumatic pains, was suddenly seized with shivering, which was followed by a slight pain between the shoulders. He became much depressed in spirits, and in the following week had several shivering fits, lost his appetite, and was feverish. He then became a patient of the Birmingham Dispensary.

When visited his countenance was flushed, his urine was thick and high coloured. He had cough followed by expectoration of thick, slate-coloured sputa. The respiration was hurried; the pulse small, frequent, and intermittent. The pulmonary murmur was rather coarse. A to-and-fro creaking was heard over the precordial region synchronous with the heart's action. He was bled to ℥xij, and strong mercurial ointment was rubbed in over the region of the heart. In a few days' time he was much relieved, and the creaking sound disappeared. In less than three weeks' time he left his bedroom, and expressed himself as being much better. His pulse had become regular, but remained very frequent. He had not, however, regained either flesh or appetite. From this time he was not heard of until on the morning of his death, which took place eight weeks after his first seizure, but he was not then seen.

Inspection. The pericardium was connected to the whole surface of the heart by cellular tissue, and could be easily dissected from it. The whole of the right lung and the upper third of the left lung were of a dark gray colour, and were studded with small tubercles, and gray granulations, and masses of gray induration, which contained patches of tubercular matter. Some of the tubercles, about as large as a pea, were softened, and many small cavities were thus formed, but there were none of a larger size.

Duration. In three cases death took place in less than five weeks from the appearance of the first symptoms; in eight about seven weeks; in seven about eight weeks; in two about nine. Besides these cases, there was one in which death occurred fourteen weeks after the first symptom; but towards the close of life the disease ran a very acute course.

Case CXVI.

Acute phthisis.

A brewer, æt. 25, fell with his left side on the edge of a barrel, and the contusion was followed by pain in that spot and a cough. The former was soon relieved by the application of leeches, but the cough remained, and was attended by dyspnœa, which gradually increased. Thirteen weeks after his fall he was admitted into the Birmingham General Hospital. He was thin, had a very anxious expression of countenance, with some lividity, very rapid and laborious breathing, and a constant, short, dry cough. The pulse was very frequent; the skin was hot, and the tongue coated. He had great thirst, but complained of no pain. The expectoration consisted of thick, clear mucus. The bowels were regular.

A large mucous rattle was heard all over the chest, but more especially under the right clavicle, where there was incomplete vocal resonance. The chest sounded clear on percussion. The dyspnœa and lividity of face increased, and he died nine days after his admission into the hospital, and fourteen weeks after his fall.

Inspection. Both lungs adhered to the chest, and in some parts of the false membrane which connected them to the ribs tubercular matter was deposited. The substance of the lungs was redder, heavier, and more friable than in its natural state, but it crepitated on pressure, and floated in water. They were universally studded with tubercles and gray granulations, some of which were invaded by yellow tubercular matter. At the posterior part of the upper lobe of the left lung there was a cavity that would have contained a walnut, but in no other part were the tubercles softened. The mucous membrane of the air-tubes was generally more vascular than natural, and was lined with muco-purulent secretion.

Small, round, opaque tubercles were found in some false membranes connecting the liver with the abdominal walls, in the liver itself, in the kidneys, and in the spleen; but in this latter organ there were also bodies resembling semi-transparent gray granulations.

The mesenteric glands were enlarged, and there was ulceration in the colon. The arachnoid membrane of the brain was thickened and very opaque in patches, there were some tubercles upon it, but no increased vascularity was observed in it.

It would appear that the disease at the upper portion of the left lung was running the usual chronic course, when suddenly it assumed the acute form. Although, therefore, this case has not been included in the twenty above alluded to, yet, strictly speaking, it falls within the class of cases now under consideration. I have observed the same thing to occur occasionally during the course of other cases of ordinary chronic phthisis.

Invasion. Dr. Stokes states, that most of the cases of this kind, which fell under his notice, came on as the sequelæ of fever, an interval existing between the crisis of the fever and the new attack. This was not observed in any of my cases, nor apparently in those of Louis; but, if those detailed in the chapter on Plastic Pneumonia be considered as cases of phthisis, then my observations will coincide with those of Dr. Stokes. In general, the period of invasion was clearly marked, which may well be seen in the following cases.

CASE CXVII.

Acute phthisis.

A shoemaker, æt. 22, an in-patient of the Birmingham General Hospital, pale and thin, had enjoyed good health until he took cold from leaving off a flannel waistcoat fourteen days before his entrance into the hospital. He first felt a sore throat, followed by a faintness, and a pain in the dorsal region, shooting up between his shoulders; a cough then set in, which gradually increased.

Examined three weeks after his first seizure, his tongue was found red and moist. He complained of a sore throat, but only slight redness of the fauces was observable. His bowels were regular. The pulse was feeble, 124. Respiration 44. Great dyspnœa existed, with much expansion of the alæ nasi. There was expectoration of clear mucus. He could lie in any position, but preferred reclining on his left side, with his head slightly raised. Percussion elicited a clear sound over the whole of the chest, except at the posterior part of the left side. Bass-viol and cooing sounds were heard all over the chest, mixed with an occasional mucous click, and accompanied by a very feeble respiratory sound. Around each nipple, more particularly the right, a sub-crepitant rattle was heard. At the lower and posterior third of the left side a mixture of dry and large moist rattles was perceived, and in one spot there was decided tubal respiration, strongly resembling metallic tinkling, with slight bronchophony. The dyspnœa gradually increased, respiration reaching 60; the other signs remained the same, except that on one occasion some traces of rusty-coloured sputa were seen. He died five weeks after his first seizure.

Inspection. Both lungs were rather heavier, and more easily torn than in their natural state. They were completely studded with gray granulations and yellow tubercles, most of which were about the size of a mustard seed; but, at the bottom of the back part of the left lung, two or three had reached the size of a pea, and were soft and primrose-coloured. For three or four inches the mucous membrane of the right bronchus and its divisions was much injected, and of a violet colour; in the smaller divisions it was extremely pale, and contained much thick mucus. In the bronchial tubes at the bottom of the left lung the lining membrane also was of a deep violet colour. There was some red hepatization here, containing a mass of pulmonary apoplexy as large as a walnut, in which were some yellow tubercles. A larger mass of pulmonary apoplexy existed in the middle of the right lung, the lower edge of which was slightly emphysematous. A little lymph was lying in thin films over the fissure of this lung. Small yellow tubercles were found in the kidneys, spleen, and mesenteric glands.

The existence of phthisis in this case, and also in Cases CXIV and CXVII, was recognised by Dr. Eccles, who formed his opinion from the general signs alone. By his permission the cases were examined, in company with Mr. William Bowman, at that time a house-pupil of the hospital, and under the same permission the notes are published.

CASE CXVIII.

Acute phthisis.

A girl, æt. 13, previously in good health, was seized with shivering, and was very feverish. A cough set in without expectoration, her breath gradually became shorter and shorter, and she began to lose flesh. She was seen in consultation with Mr. Thomas Freer five weeks after her first seizure. She was then labouring under extreme dyspnœa, with respiration 48 in the minute; the alæ nasi expanded with every breath, and the lips were blue; but the face was pallid and haggard. Her pulse was 130. The cough was very troublesome and almost incessant, accompanied by clear and very scanty expectoration. Slight dullness was perceived on percussion under the left clavicle. A mixture of cooing sounds and moist rattles was heard all over the chest, in some parts large, and in others rather fine. Where the respiratory sound could be detected it was very coarse. No remedies seemed to relieve her for any length of time, and she survived fourteen days, dying suffocated, the expectoration remaining clear and becoming more adhesive.

Inspection. The whole of the left lung adhered closely to the thorax, but the adhesion was torn through without the lung being lacerated. The lining membrane of the bronchial tubes was generally rather redder than usual, and in some spots livid and softened.

Both lungs were thickly studded with yellow tubercles of the size of a pin's head, and a few gray granulations. These latter were clustered thickly and mixed with yellow tubercle near the apex of the left lung, and formed gray induration. The parenchyma of the lungs was engorged, and heavier than usual, from the presence of tubercles and granulations rather than from the amount of congestion. It crepitated, and was not very easily broken down.

In other cases the exact period of invasion could not be determined as in this case.

Case CXIX.

Acute phthisis.

A girl, æt. 6, had complained for some weeks of a stitch in the right side, and was troubled with a short dry cough. She was rather emaciated, and had an anxious expression of countenance. The pulse was small and frequent, and the respiration hurried.

There was some little dullness on percussion at the bottom of the right side. Cooing sounds were heard all over the chest, and a loud, to-and-fro rubbing sound over the greater part of the right side. In ten days' time she was again seen in consultation with Mr. Allarton. Her breathing was then most laborious and distressing, and her face was pinched and anxious, her lips being purple, and the alæ nasi expanded. She was very thin. Her pulse was frequent and small. The expectoration varied, at one time consisting of clear mucus, and at another being muco-purulent. In addition to the cooing which was heard over the chest, a fine crepitation was audible in many parts. Percussion elicited no dullness of sound. She died in less than three weeks.

Inspection. The right lung was connected to the chest by very strong bands, and the costal pleura was injected. The whole of the right lung was studded with gray granulations and opaque tubercles. Some of the granulations were reddish and soft, others gray, hard, and semi-transparent, and in a few there were yellow specks either at the centre or sides. Some of the yellow opaque tubercles had reached the size of a rape seed, and were soft. Towards the base of the lung there were a few patches of agglomerated granulations, presenting the appearance of what is generally allowed to be the result of chronic pneumonia. The pulmonary structure crepitated, but was reddish and heavy. The upper lobe of the left lung was of a pale colour, from its containing some groups of aggregated granulations and some large softened tubercles. Through the lower lobe granulations and tubercles were scattered, but not so thickly as elsewhere. The mucous membrane of the large air-tubes was swollen and livid, and of the smaller ones very pale. There was much muco-purulent secretion in the tubes.

There are two prominent features observable in the general signs in these cases. There was such an assemblage of them as is observed in fever with bronchitis, viz., thirst, anorexia, heat of skin, frequent pulse, &c. At the same time there was a remarkable amount of dyspnœa in every case, and in most of them a lividity of countenance not observable in ordinary cases of bronchitic fever, but rather such as is seen in very acute attacks of bronchitis. The sputa, however, were not viscid, and the cough was often slight at the onset; facts inconsistent with the existence of acute bronchitis.

In Dr. Stokes's cases the expectoration is described as scanty, viscid, and often tinged with blood. A slight appearance of pneumonic sputa was only once observed in the cases seen by me.

In most of them pain was felt in the sides and back of the chest.

There was not hemoptysis in a single case.

The ribs were never immovable.

There were no venous pulsations of the neck, dropsy, nor any other general signs of obstruction of the circulation at the heart.

The general symptoms, therefore, indicated a high state of fever, and very considerable pulmonary oppression which could not be produced by the slight amount of pleurisy, bronchitis, or pneumonia present.

In nearly every case percussion elicited a clear sound over the greater part of the chest from first to last. In the following case, however, the chest was observed by Mr. W. C. Freer to give out a duller sound than usual.

Case CXX.

Acute phthisis.

A farming-lad, æt. 12, had been poorly fed and hard-worked from an early age. Five months ago he felt slight tenderness over the back-bone, in the loins, and experienced some little difficulty in moving. One month since he was thrown violently to the ground, immediately after which the tenderness on the spine increased very much. When admitted into the Birmingham General Hospital there was found to be backward curvature of the spine at the seventh dorsal vertebra, over which there was great tenderness on pressure; and in this spot he constantly felt rather severe pain shooting up between the shoulders. The power of his lower extremities was somewhat impaired. He was emaciated, and he laboured under a considerable amount of fever, with great thirst and heat of skin. His pulse was 120, small. Dyspnœa was very urgent. His hands and face were of a livid colour. He had cough, with slight expectoration of clear ropy mucus, which had existed for some months, but had attracted little notice. Respiration was 44 in the minute. The chest generally sounded duller on percussion than usual, but not in one place more than another, nor more so than in some persons in health. Sub-crepitant rattle, with bass-viol and cooing sounds, and in some places with creaking very near the ear, were heard more or less over the whole surface of the chest, but stronger under the clavicles, where vocal resonance was detected. He died in six days, without any coloured expectoration, apparently suffocated.

Inspection. Both lungs adhered more or less closely to the chest, and were studded with yellow unsoftened tubercles and gray granulations, many of which were becoming yellow and opaque. Some tubercles at the summits were larger and softer than others, but there was no cavity. The body of the ninth dorsal vertebra was absorbed, and a mass of cheesy scrofulous matter occupied its place.

In two other cases also the sound seemed to become progressively duller; but in no case was this sign strikingly marked.

Pneumonia was not indicated by a mixture of pure crepitation and dullness on percussion, succeeded by absence of respiratory sound, except in one spot in Case CXVII. Nor was intense bronchitis proved to exist by extensive and small mucous rattles. Bass-viol and cooing sounds were frequent, indicating a congested rather than a highly inflamed state of the mucous membrane; and the moist rattles were generally confined to certain spots. There were no evidences in any one case of the blood having been obstructed at the heart.

The physical signs were all therefore of a negative character, showing that none of the usual causes which produce obstruction of the blood in the lungs were present in these cases.

Such being the fact, it may be argued that the dullness observed in the three cases above mentioned, not arising from the ordinary causes, must have depended on this unusual cause, the rapid increase of solid matter in the lungs in the shape of granulations and tubercles; and such doubtless was the case. If, therefore, this sign were of frequent occurrence, it would be of great value as diagnostic of the disease in question. As such it has been brought forward by Dr. Stokes, and in his experience this sign is seldom absent, and is not difficult of discovery. It was only perceived, however, and that most faintly in three of the twenty cases of acute phthisis and gray granulation; nor does it appear to have been observed more frequently by Louis. This is easily accounted for. Supposing the sonoriety of the chest to be considerably diminished, it may be very difficult of appreciation. We have in truth here no parts of the chest to compare with each other; for not only are both sides similarly affected, but also every part of each side; the value of differential examination, therefore, so strongly insisted upon by Dr. Stokes, is here altogether lost to us. The only comparison we can make is that of time, which requires that we should carry in our memory the exact amount of sound emitted from the chest on percussion at a previous examination. The difference may sometimes be appreciated, but I believe it will very seldom be discovered even by the most practised ear. This sign may possibly possess some value in the hands of so skilful an auscultator as Dr. Stokes is well known to be, but as a guide to the bulk of the profession it must be comparatively useless.

Now although there are no positive signs by which we are enabled accurately to discover the nature of this disease, yet it is clearly ascertainable from the results furnished by negative signs, *par la voie d'exclusion.* It is certain that there is a sufficient cause for the production of great obstruction of the blood in the lungs, in the rapid development of numerous granulations and tubercles in them; if, therefore, evidences of such obstruction exist in all these cases, and if the signs, both general and physical, prove that no other sufficient cause is present, that, and that alone, remains as the cause, and thus the disease is most clearly made out.

It is submitted therefore that, when symptoms of high fever and great pulmonary obstruction are observed unaccompanied by signs of acute bronchitis, pleurisy, pneumonia, or heart affection, the disease may safely be pronounced to be *acute phthisis* or vesicular pneumonia.

CHAPTER XXII.

TREATMENT OF PHTHISIS PULMONALIS.

Various means have been proposed for arresting the progress of phthisis pulmonalis; and these have consisted of *specific remedies*, or certain *plans* of treatment.

None of the specific remedies which have been in past years so zealously advocated and supported by numerous cases of apparent cure, have stood the searching scrutiny of the numerical system of investigation as conducted by Louis.

More recently two others, *naphtha*[1] and *cod-liver oil*[2] have been proposed. I have thought it right, therefore, to test these remedies in a similar manner, by making trial of each in one hundred cases of phthisis.

Naphtha. In one half of the cases, cavities already existed in one or both lungs; and, in the other half, there were evidences of solid deposit in the summit of one lung or both. In the former half no permanent benefit was derived from the administration of naphtha. Forty-four terminated fatally, and the remaining six present all the signs of advanced disease. In the latter half, this medicine seemed in some cases to improve the tone of the digestive organs, more especially of the stomach when it was irritable; but, on the whole, the progress of the disease was not arrested in any one case; and on comparing these fifty cases with fifty others, tubercular softening was found to have come on as rapidly in one class of cases as in the other; so that, in my hands, naphtha has not been found either a specific remedy for phthisis, or one of any great value.

Cod-liver oil. The ratio of the incipient to the confirmed cases was the same as in the last-mentioned trial. Twelve of the incipient cases were decidedly improved. Of the confirmed cases six were greatly relieved. Of these, four are now alive, and two, of which Case CVII is one, are dead. Of the four who survive, two are strong and plump, and only expectorate pearly mucus. The other two have the disease in a very chronic form. The whole six were greatly emaciated, and were rapidly sinking when they commenced taking the oil. They all soon gained flesh. In five cases of the 100, it was obstinately rejected by the stomach. In eleven it purged, so that it could not be borne in sixteen cases. In two or three cases, after acting beneficially for some time, it purged; this took place in Case CVII, but its good effects remained. Having found such desirable and unlooked-for results, I continue to prescribe it, and to watch its action. All that can at present be deduced from this trial is, that although cod-liver oil is not a specific remedy for phthisis, its administration has been found useful in certain cases, when combined with a judicious system of regimen and diet.

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[1] Dr. Hastings, on Consumption.

[2] Dr. Hughes Bennett, on Cod Liver Oil; Dr. Péyreya, of Bordeaux, on Pulmonary Consumption.

Since the above was written, I have witnessed very beneficial effects from the use of this medicine in several other cases.

Plans of treatment. Three plans of treatment have been employed, and these may be denominated the *antiphlogistic,* the *expectant,* and the *tonic.*

The *antiphlogistic* plan was strongly advocated by Broussais, and subsequently by Dr. Stokes,[1] who endeavours to show that it is "the true mode of arresting the disease in its early periods." In carrying out this plan, venesection is more or less freely employed at the onset of the disease, and is followed by local depletion and counter-irritation, and in some cases by the administration of mercury. This treatment is evidently based on the belief, that tubercle in the lung chiefly results from acute inflammatory attacks of that organ; and its principal object is, therefore, to subdue and remove the local cause.

In the *expectant* treatment, venesection is more sparingly employed, but leeches and counter-irritation are occasionally used. Its chief characteristic lies in the means which are taken for preventing exposure to cold. "It was formerly thought," writes Dr. Graves,[2] "that consumption arose from inflammation of the lung, and on this erroneous reasoning was founded its preventive treatment. The patient was confined to his room, and kept in an equable temperature wrapped up in flannel." The diet is frequently restricted to milk, slops, and farinaceous food; and expectorant medicines are administered, often with the effect of nauseating the stomach and impairing the appetite. This treatment is, like the last, principally directed against irritation of the lung; it is employed by a large class of medical practitioners.

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[1] Diseases of the Chest, p. 440.

[2] Dr. Graves's Clinical Medicine, p. 291.

The *tonic* treatment is advocated by Louis, Sir J. Clark, Dr. Graves, Dr. C. B. Williams, and others, and is well described in the words of Louis.[1] "Regularity of habit, a just appropriation of time to study, and exercise of the body, occupation of well-ventilated apartments, with good exposure, succulent diet, the use of bitters and chalybeates, and cold bathing, are what we should recommend to persons of all ages, more especially to those who are of lymphatic temperament, and whose relations have fallen victims to phthisis." This treatment he carries more or less through the different periods of the disease; occasionally, however, meeting an attack of acute inflammation or hemoptysis by antiphlogistic measures.

Thus the principal object of this treatment is directed to the improvement of the constitution generally, and through it to the removal of tubercular cachexia; at the same time, the irritation of the lung is not overlooked, but is for the most part treated with sedative medicines, and very moderate local depletion; by some of its advocates also with counter-irritation, and by others with mercury in certain cases. But these remedies are so selected and administered, as not materially to lower the system, and thereby interfere with the primary object of the treatment.

It would be very desirable to test the value of these different plans of treatment by the results of practical experience, but at present this is impossible. Even Louis has been unable to collect the materials necessary for this purpose; and, for my part, although I have not hesitated to make trial of a particular medicine or remedy in a certain number of cases, when I had reason to believe that no injurious effect would follow its administration; yet I could never bring myself to employ plans of treatment so little in harmony with the nature and origin of the disease, as I consider two of those under consideration to be.

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[1] Op. cit. p. 541.

I have certainly had numerous opportunities in public and private practice of witnessing the effects of all these plans of treatment in the hands of others, and could adduce striking instances of the benefit of a change from the antiphlogistic and expectant to the tonic treatment, and could also contrast the effect of different plans of treatment on members of the same family; but as so few are ever relieved, I do not think that the analysis of any number of cases which have come under the observation of one individual could afford sufficient data for the induction of satisfactory conclusions. With Louis, therefore, I am content to seek for rules of treatment solely in the nature and causes of the disease, as far as they are at present known to us.

Our inquiry into the nature and development of phthisis has shown, that the deposition of tubercle in the lung is invariably preceded by an altered state of the constitution generally. It is therefore clearly deducible from this, that the principal object of treatment should be the improvement of the constitution, and that the means selected for allaying the irritation of the lung should be of such a nature as not materially to interfere with this the primary object.

It has also been seen that this constitutional derangement is of an asthenic character, a kind of chronic typhoid state. It is true that, in a certain stage of the disease, the condition of the blood resembles that which is found in inflammatory affections; but it must be remembered, that such inflammations are as frequently of an asthenic as of a sthenic character. This state of the blood, therefore, does not call for antiphlogistic measures.

The nature of the disease, then, as far as we are acquainted with it, indicates the invigoration of the system by nutritious and tonic regimen and diet, as the best means of fulfilling the primary object of treatment, the correction of the tubercular diathesis.

The known causes of the disease point to the same course. Age, sex, and hereditary influence, inasmuch as they cannot be removed or modified, suggest no treatment; but the general causes which predispose to phthisis, in common with many other chronic diseases, are one and all of a nature to induce debility of the system; and the same may be said of the special causes, moral and physical depression, febrile attacks, derangement of the functions of the uterus, &c. &c., and consequently they can only be removed or modified by tonic treatment.

But practical experience teaches us that with debility of the frame is associated a certain amount of irritability that demands especial attention, and without which all attempts to strengthen the system will often fail. The same experience teaches us that this irritability is best removed by sedative remedies, which happily are equally well calculated to diminish the special irritation of the lung, and which also are so far from interfering with the tonic treatment indicated, that their administration harmonizes with it, and greatly assists in forwarding the object proposed by it.

The nature and known causes, therefore, of phthisis pulmonalis suggest a grand principle on which to proceed in our endeavours to arrest its progress. *The union of tonic and sedative remedies.* To this principle the antiphlogistic and the expectant plans of treatment are opposed. They rest on a false foundation, because they assume that inflammation of the thoracic viscera frequently induces phthisis in persons previously healthy, because the correction of tubercular diathesis is not the principal object to which they are directed, and because their tendency is to lower the system, and thereby to favour the constitutional taint. They are consequently counter-indicated by the nature and known causes of the disease.

In applying the principle here deduced to the *prevention, removal,* and *palliation* of phthisis, it will be found that circumstances will occasionally arise, which demand that the plan of treatment should be to a certain extent modified; nor does its adoption prevent our employing counter-irritation at the same time, because this remedy may be so managed as not materially to lower the patient. Admitting Louis to be right in condemning its indiscriminate use as a preventive of phthisis, I cannot be prevailed upon to neglect this remedy, which I have found of the greatest service in the incipient stage of the disease; and which I have tried too extensively to be deceived in my estimate of its value. Dr. Graves may possibly claim too much for this plan, but Louis, according to my experience, attaches too little value to it.

Prevention. It is impossible to commence the prophylactic treatment of phthisis at too early an age, because, if it is conducted on sound principles, it tends to strengthen and invigorate the constitution, whether there be an hereditary disposition to the disease or not. If the complaint has existed in the mother's family, or if she should be in a delicate state of health, a wet-nurse should, if possible, be provided, and maintained in good health by nutritious but not over-stimulating diet, by regular exercise in the open air, by frequent ablutions of the body, and by the habitation of large and well-aired apartments. During the time the infant is at the breast, should it not acquire the average degree of plumpness, or should its flesh be pale and flabby, it should have a few teaspoonfuls of meat-tea daily. A strong prejudice against this kind of food exists in the minds of many medical men, parents, and nurses. I can assure them that it is without foundation. When at a later period children are gorged with large quantities of half-masticated solid meat, serious mischief may arise; but I have never seen any evil effects result from a judicious administration of meat-tea; whilst under its use alone I have seen infants restored from a fearful state of marasmus to vigorous health.

Nor can children be too early accustomed to the open air, without which the best food and nursing will fail to rear them in health and strength. There are few days in the year in which they cannot be carried out, and these are when fog and keen east winds prevail. When they have become debilitated from attacks of illness or any other cause, nothing will so quickly restore them to health as abundance of fine fresh air at a little distance from their usual place of abode. The clothing should be moderately warm, thin flannel being next the skin, and so arranged as to leave the body unconstrained, and to allow of the free movement of all its parts. The surface of the body, more especially the chest, should be well washed and rubbed, and although it may be advisable at certain seasons to take the chill off the water, that which is applied to the head should be always cold; because congestion, followed by tubercular deposit, is apt to occur in the brain and its membranes during early youth, and nothing tends more to prevent congestion of the head than cold affusion.

It is really surprising how seldom this plan is adhered to, simple and rational as it is, and applicable to all ages. This is partly owing to the advice of medical men who are advocates of the expectant treatment, but more frequently to the prejudices which exist in the minds of parents and nurses. They naturally enough consider phthisis as an exaggerated "cold of the lungs," and think that its prevention must necessarily consist for the most part in precautions against catching cold. It not unfrequently happens that from a blameable timidity, their medical attendant allows them to act in accordance with their prejudices, instead of insisting on that rational mode of treatment which he knows to be suitable and proper.

When the period of dentition approaches, restlessness and other symptoms of cerebral disturbance often manifest themselves, and are frequently treated as if they were indicative of active congestion or inflammation of the brain. Such is doubtless the case sometimes, but oftener the brain merely sympathises with the nervous irritability of the system, which may be relieved by mild sedative medicines, whilst the primary cause is removed by favouring the passage of the teeth by incision of the gums. A few drops of syrup of poppies or of morphia, occasionally administered, often produce the most beneficial effects not only at this period, but later in life, when restlessness and irritability are observed without any manifest derangement of the important functions of the body. Louis has shown the groundlessness of the dread entertained by many of sedative medicines producing congestion of the brain, or arresting the development of the intellectual faculties of infants.

We must not be content with merely thus endeavouring, by every means in our power, to invigorate the constitution during infancy and youth, but we must also be alive to the faintest indications of a tendency to tubercular deposit in any part of the body. It is true that, at this period of life, tubercles are not usually found in any great quantities in the lungs, but they are frequently engendered in the brain and its membranes, in the peritoneum, and in the glands of the mesentery and of the neck.

The symptoms of tubercular deposit in the brain are often extremely obscure; when, however, evidences of meningitis are unequivocally manifested, they must be met with proper energy. I have seldom found it desirable to take blood, and have often seen a fatal prostration follow the application of a very few leeches. Purging by calomel has appeared to me to be the most successful practice in such cases, assisted by mercurial frictions, if the symptoms run very high.

Tubercular peritonitis will often be much relieved by mercurial and opium frictions, combined with mild nutritive diet, so prepared as to be easily digested. I have seen several cases thus completely cured.

Tuberculization of the glands of the mesentery is very frequently a sequela of that low form of gastro-enteritis with which children are so much troubled; and it has appeared to me to have been produced rather by the treatment than by the disease in many cases. It is true that these glands become congested during ulceration of the intestine in typhus fever, and the same may take place in ordinary gastro-enteritis, but if the system be reduced by purging with calomel, jalap, and scammony, and by low diet, the tubercular diathesis may become established, and the congestion may be succeeded by tuberculous matter, or the typhic deposit may be converted into it. A more rational treatment consists in the administration of alkaline and alterative medicines with some mild sedative, and of a combination of farinaceous food with the liquid parts of animal flesh, whereby a material is offered to the organs of digestion which can be very easily converted into healthy blood. When mesenteric disease has really set in, much benefit will often be derived from iodide of potassium, and sometimes from cod-liver oil.[1]

Nor must we lose sight of this grand principle of treatment here advocated in combating the more acute attacks of childhood and youth; employing antiphlogistic measures as sparingly as possible, and trusting rather to mercury and antimony, according to the nature of the attack. It is altogether a mistake to suppose that children are injuriously depressed by antimony, if they are not at the same time lowered by other active remedies.

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[1] Potassii Iodidi . . gr. xij.
Liquoris Potassæ (*Brandeish*) ʒiv.
Aquæ destillatæ . ℥iss. Misce.
Sumat ʒj vel ʒij, cum cyatho aquæ ter die.

When the age of puberty is approaching, our attention must be directed to the generative system. In the female we have to watch and regulate the development of the uterine functions, a point of great importance, as will be seen in the sequel. In the male we must look most closely to the nervous system, prepared, if necessary, to cast away all false delicacy or fear of giving offence, and to point out the pernicious effects of masturbation, which too often destroys the health, and paves the way for the development of phthisis.

At all ages, too, we must strive to prevent inordinate application to study and sedentary occupation, and to promote regularity of out-door exercise and mental repose after any extraordinary calls have been made upon the mind. How many a youth of high promise has been sacrificed at the altar of ambition, and fallen the victim of phthisis with the wreaths of intellectual conquest yet unwithered on his brow! Case cv furnishes a melancholy instance of this. Never has the warning voice of the physician been more required than at the present moment, when the race for literary and scientific distinction is so hotly contested.

The importance of out-door exercise cannot be overrated. It not only tends to circulate the blood freely and thereby conduces to the general health, but it also tends to prevent congestion of the internal organs, and amongst them the lungs; so that it is a preventive of both causes of phthisis. The evils arising from the respiration of cold air have, in my opinion, been much exaggerated; for determination of blood is much more likely to be produced by cold on the surface of the body, whereby the blood is driven from the external capillaries, than by its effects on the interior of the air-passages. When cattle suffer from inflammation of the thoracic viscera they are immediately allowed an abundance of fresh air, whilst the surface of the body is warmly clothed. So, too, we do not find that thoracic inflammation more frequently supervenes on other diseases, or runs a more severe course in the wards of our hospitals which are well ventilated and supplied plentifully with fresh air, than in those private houses in which the external air is most carefully excluded from the sick room. After a long confinement to the house the use of the respirator may be valuable for a short time when the patient leaves it, should the weather be severe; but I have never seen any advantageous results from its habitual use in phthisis.

If these prophylactic measures are carefully carried out, the digestive organs will in general duly perform their office, but the slightest derangement in their functions should be carefully watched; because the disease has been seen to be essentially one of abnormal nutrition. It would be useless to enter into the minutiæ of the treatment required in this respect, which must vary with individual cases, but which may be carried on in strict subservience to the outlines of the plan here advocated. One of the most frequent effects of such derangement is acidity of the alimentary canal; and this may be temporarily remedied by the use of fluid magnesia after the first process of digestion has been accomplished. If, however, the diet and habits of exercise be carefully regulated, but little medicine will generally be required.

It is really surprising how seldom phthisis shows itself in persons who have been uniformly subjected to this prophylactic treatment; a remark, the truth of which is confirmed by statistical researches on the predisposing causes of the disease, which show that the tubercular diathesis is very frequently induced by bad management, and consequently may be much oftener prevented than has hitherto been supposed.

Removal and palliation. We have now to consider by what means we shall meet the approaches of the disease in its various forms, and treat it during its course when it has once set in.

The invasion of phthisis may occur in a latent form, or it may be ushered in by symptoms of irritation of the bronchial tubes, of the trachea and larynx, or by hemoptysis; or else it may take place in the acute, by some called, the inflammatory form.

Invasion by bronchitis. The first thing observed is a short cough, for some time unaccompanied by any expectoration; and when this does appear, it is clear and frothy. A sense of weariness and languor succeeds, and sooner or later flushes and heat are felt towards the evening. Slight flying pains about the chest and shoulders are also complained of sometimes. In many cases emaciation then takes place. The pulse is generally increased in frequency. Sometimes auscultation reveals nothing; more frequently, however, as this state of things has generally existed for some time before medical advice is sought, the pulmonary sound under one clavicle is found to be coarse and prolonged during expiration, and this is soon succeeded by cooing sounds, or an occasional click. It is often a long time before any dullness is perceived on percussion.

Now there is nothing in all these symptoms to counter-indicate the constitutional treatment, and therefore mild tonic medicines and regimen are required; out-door exercise, change of air, nutritive but not stimulating diet, &c., may be ordered. But as it is clear that bronchial irritation exists, it must, if possible, be allayed by mild sedative medicines, and by promoting secretions from the mucous membrane of the air-tubes, and from the skin. The compound cascarilla mixture and a small blister on the chest are remedies suitable to this purpose, and are often attended with the best results, our object being attained without nauseating the stomach. This failing, and there still being no high fever, a more stimulating plan will often succeed, such as the administration of ammoniacum mixture, with tincture of squills, naphtha, nitric ether, and paregoric, or a morphia linctus.

The flying pains in the chest are often much relieved either by the soap and opium liniment, or, if obstinate, by mercurial and opium friction, as in pleuritis. In fact, when the ear is placed on the chest, it sooner or later detects the creaking or rubbing sound indicative of a low form of plastic pleuritis, which gives rise to these pains and to the adhesions between the pleural surfaces in phthisis.

Latent form. Cough is seldom present in the earliest stage of this form of incipient phthisis. The first symptom complained of by the patient is a tightness of the chest, with dyspnœa on exertion. At a later period cough sets in, and is often very troublesome and distressing; and other signs are also added, which have been mentioned as occurring in bronchial irritation, and then the two varieties cannot be distinguished the one from the other by general signs.

The difference between them, however, is chiefly marked by the physical signs. When the chest is examined at a very early period, before the patient has complained of any uneasy sensation in that part of the body, feebleness of the pulmonary sound in some spot under one clavicle is observed, and even then some dullness is found on percussion. In a short time, either the pulmonary sound ceases in this spot, or becomes very coarse and bronchial, and prolonged during expiration, and buzzing bronchophony makes its appearance; at the same time the dullness on percussion is increased, but still there may be neither dry nor moist rattles.

This form of invasion is most frequently found in females, or in males of a decidedly lymphatic temperament. Preceded by an anemic state of the system, and the suppression of the menstrual discharge, it is one of the most common forms of the invasion of phthisis, and one on which it is very desirable to fix the attention of young practitioners, because it is very frequently overlooked; and, also, because when discovered in time, and treated with judgment, the disease is very often arrested. It was found to have commenced thus in nearly 500 females out of 800, in whose cases the symptoms of invasion were accurately traced, and who were from 16 to 28 years of age.

I have found expectorant medicine of no use, indeed worse than useless, in this form, in which tonic treatment is required in its full extent. Some preparation of iron may generally be given, with an amount of sedative medicine proportioned to the degree of irritability existing. But, above all things, horse-exercise and change of air are most valuable, joined to a generous meat-diet, with a tolerably free use of porter or bitter beer, and friction of the surface of the body; at the same time flying blisters of small size may be applied to the chest, a crop of pustules brought out by tartarized antimony or croton oil, or redness may be produced by turpentine. In some cases the blister may be dressed with mercurial ointment.

When the uterine functions are suspended, every effort must be made to restore them by the administration of iron and aloes, and the use of the hip-bath, joined to the means just detailed; and these failing, by leeches applied to the genitals, if the strength be not greatly impaired.

These remarks only apply to the invasion of the disease;

when the menstrual discharge has been suppressed in an advanced stage of phthisis, it would be most injudicious to endeavour to force it, and thus bring on a drain upon an almost exhausted system.

The palpitation of the heart, which often accompanies this state of the health, is generally relieved by belladonna frictions over the precordial region.

It may be necessary occasionally to relax and modify this treatment, but it is applicable to the great majority of cases, and in numerous instances has been attended with the most beneficial results.

Invasion by hemoptysis. Sometimes an attack of hemoptysis is the first symptom observed. Many practitioners invariably treat this formidable symptom by venesection. Even Louis remarks, that we can scarcely avoid having recourse to it when the patient retains a certain amount of strength and flesh. The form of expression used, however, implies a doubt in the mind of this sound physician. Experience has led me to demur even to the exceptional case thus put, and to refrain from venesection whenever there is a suspicion of phthisis. The following was the last case of the kind in which I recommended it.

Case CXXI.

Hemoptysis—Phthisis.

A lady, æt. 50, had suffered for some months from cough, with slight muco-purulent expectoration, and had presented physical signs of solid deposit in the summit of one lung. She was suddenly seized with a violent attack of hemoptysis. Seen in consultation with Mr. Langston Parker, her face was flushed and her eyes bloodshot. She laboured under great dyspnœa. The pulse was remarkably full, hard, and sharp, 100. There was some dullness at the upper part of the left lung, with absence of respiratory murmur over a small space. The action of the heart was strong and quick. Twelve ounces of blood were taken from the arm with great relief. Tubercular softening, however, very speedily took place, and she sank rapidly.

If ever there was a case in which venesection would seem to have been indicated it was this, and yet its effects must be considered as having been far from satisfactory. Since then two other exactly similar cases have been treated without venesection, and the results have been much more favorable.

When the attack is slight, perfect repose of body and mind, with cold acidulated drinks, will usually suffice to arrest it; but when the hemoptysis is considerable, ice and refrigerating lotions may be assiduously applied to the chest, and opium and acetate of lead freely administered; at the same time the blood may be solicited towards the lower extremities.

Now if venesection be not necessary at the outset of the disease, surely it is counter-indicated after hemoptysis has appeared in its later stages, when the strength of the patient must be necessarily diminished.

Invasion by tracheal irritation. Sometimes phthisis is preceded by unequivocal signs of tracheal and laryngeal irritation. There is a ringing cough, with soreness and pain in the trachea, hoarseness of voice, which is soon followed by fever and emaciation; and yet nothing is at first discovered in the lungs by auscultation. No treatment avails in this form of invasion, according to my experience. We may leech, blister, mercurialize, and order inhalations of all kinds of vapours, sometimes with the effect of relieving the patient for a day or two at the most; but I have never known any improvement beyond this, and therefore soothing treatment seems all that we are justified in employing.

Febrile invasion. When phthisis succeeds to typhus fever, we may treat it on the principles already laid down; but it is of the greatest importance to prevent its development if possible, and this we can only expect to do by guarding against the prostration of the vital power which occurs in typhus, and which lays the foundation of phthisis.

I have long been in the habit of anticipating what is denominated the turn in all febrile attacks, when the signs of extreme debility become evident. Listening carefully to the systolic sound of the heart, as recommended by Dr. Stokes, and thus seeking the earliest indication of prostration, I have been accustomed, on finding it, at once to commence the administration of quinine, wine, or ammonia, by cautious doses, irrespective of the state of any other organ, or of any other symptom. I have never had occasion to regret this line of treatment either in typhus or in the exanthemata. The strength has been very quickly restored in this manner, and phthisis has seldom supervened.

The *acute form of invasion*, as illustrated by the cases in Chapter XXI, demands prompt and energetic measures. We cannot trust to the constitutional treatment alone, for unless the progress of tuberculization and of plastic vesicular pneumonia be quickly arrested, the patient will die of suffocation. When the dyspnœa is urgent and the fever is running high, we can hardly refrain from the use of the lancet, but even here I have seldom employed it, because experience has taught me that the most acute inflammation of the thoracic viscera may be often successfully treated without it.

If we abstain from bloodletting, then we must have recourse to mercurialization, and for this practice we have strong grounds; since a glance at the progress of acute phthisis shows that the gray granulations materially hasten the fatal termination by clogging up the lungs before tuberculization has taken place to any great extent. Now mercury is our sheet-anchor in promoting the absorption of plastic lymph. In the following case this treatment was adopted.

Case CXXII.

Acute phthisis, or plastic vesicular pneumonia, treated with mercury.

A servant girl, æt. 20, had enjoyed good health until a month before she was admitted into the Birmingham General Hospital. She was at that time in good flesh. She first felt a pain in her head and back. Ten days ago she felt rather chilly and sick at the stomach, and found her legs to "fail her." This was followed by copious perspiration, which afforded her relief. Every day towards afternoon this symptom appeared, she lost her appetite, and vomited a little occasionally. At the same time her breath became short. When examined she complained of tightness across the chest and pain under the left hypochondrium, lassitude, occasional nausea, constipation, chills and heats, loss of appetite, and intense thirst, but she had no cough or expectoration. Respiration was 40. The pulse was 110, rather small. Careful auscultation failed to discover anything abnormal in the chest, except cooing and bass-viol sounds. In a few days' time slight cough appeared, accompanied by very scanty and clear expectoration. The fever increased greatly, the dyspnœa was urgent, and the face rather livid. The respiration was 48, and the pulse 150. The bowels were now open, but there was no appetite, and but little sleep. The chest still sounded clear on percussion. Bass-viol and cooing sounds were heard all over the chest. There was no vocal resonance.

Strong mercurial ointment with camphor and opium was rubbed on the chest every four hours. In three days' time the gums were affected. The respiration fell to 43, and the pulse to 140. The fever was very much diminished, and dyspnœa much less distressing. The expectoration now became semi-opaque and yellowish, but still small in quantity. Her appetite began slightly to improve. In three weeks' time she was tolerably well, and in another fortnight the catamenia reappeared. She has remained well ever since.

As the disease advances, certain modifications in the plan of treatment here suggested will be required, in order to meet the various accidental complications which may occur in its course. But we can still only hope to remove the disease by improving the general health, and allaying the local irritation by tonic and sedative remedies; and even when all hope of removal is gone, the increasing debility of the patient necessarily calls for the same line of treatment.

Thus acute inflammations must be treated as adynamic in their character. For instance, whilst antimony is administered in pneumonia, quinine or ammonia may be given at the same time.

When subacute gastro-enteritis exists, it often gives rise to symptoms of a very formidable nature, vomiting and diarrhœa.

Vomiting will generally be best relieved by iced drinks, sometimes by solid opium, at other times by liquor potassæ, hydrocyanic acid, or naphtha; but it is often very obstinate, and thus our main dependence is taken from us, for we cannot hope to improve the blood and strike at the root of the disease as long as the greater part of the materials for nutrition are rejected from the stomach.

Diarrhœa, too, is frequently very obstinate, especially when in the later stage of the disease it is produced by ulceration of the intestines. Starch and anodyne injections have usually produced more benefit than any other means which I have used. Often, however, when sedative and astringent medicines fail, they will restrain the diarrhœa when accompanied with a tonic, such as compound kino powder with quinine.

In respect to cough and night perspirations, I have no remedy to propose but what must be well known to almost every practitioner. The cough is relieved by morphia, in doses increasing with the duration and urgency of the symptoms; and, when convulsive, is occasionally much restrained by the addition of small doses of hydrocyanic acid. When it is accompanied by excessive secretion from the bronchial tubes, the decoction of senega polygala is often very serviceable. When night perspirations are excessive, and are uncontrolled by other remedies, I have seen advantage derived from tannin.

The dyspnœa, when very distressing towards the close of life, is sometimes greatly relieved by nitric ether and sal volatile.

Little has been said of inhalation, because I have not found much benefit to arise from it, although I have tried it extensively in the manner recommended by its advocates.

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